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A BIOARCHAEOLOGICAL STUDY OF HEALTH IN THE PREHISTORIC POPULATION FROM CA-ALA-329

A Thesis

Presented to

The Faculty of the Interdisciplinary Studies Program

San Jose State University

In Partial Fulfillment

of the Requirements for the Degree

Master of Arts

by

Irina Nechayev

May 2007

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ABSTRACT

A BIOARCHAEOLOGICAL STUDY OF HEALTH IN THE PREHISTORIC POPULATION FROM CA-ALA-329

By Irina Nechayev

This thesis assesses health in the prehistoric population from the CA-Ala-329 site (San Francisco Bay area) using osseous markers of physiological stress as investigative tools. Archaeological studies from the region provide information on increase in sedentism, population growth, and depression of food resources in late prehistory. The present study examines health consequences of changing environmental and cultural conditions that occurred during a span of almost 2000 years (300 BC– AD1800).

The research, based on a skeletal population of approximately 300 individuals, reveals that overall load of stresses did not change over time. Males and females were generally equally affected by stresses. In the last prehistoric period, however, females exhibited a higher load of nutritional stresses than males. Observed skeletal markers indicate that early childhood stresses from infectious diseases and inadequate nutrient supply, which resulted in enamel hypoplasia and cribra orbitalia lesions, adversely influenced stature and longevity of affected individuals.

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CHAPTER 1: INTRODUCTION

Past human populations continuously faced the challenge of changing environmental conditions. Adaptative processes to meet new requirements for survival were accompanied by multiple stressors related to nutritional quality and availability, climatic fluctuations, social stratification, changing pattern of infectious diseases, and various other factors, which had impacts on the health status of individuals and populations. Ultimately, the assessment of health presents a valuable source of information for understanding human adaptation and in reconstructing lifestyles of past civilizations.

In application to Central California prehistory, an examination of the health of the prehistoric inhabitants of the Ryan Mound (CA-Ala-329) provides a bioarchaeological perspective on the rise and development of the indigenous populations. The exploitation of the mound continued for approximately two thousand years. Archaeological data are providing evidence of considerable social, cultural, and economic development in the region during this period. The present study investigates the health consequences of archaeologically observed events and assesses the population's adaptation to changing environmental and cultural conditions.

Health Assessment from Skeletal Remains

Health, according to the World Health Organization, is defined as "a state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity" (Gerstman 2003). However, the loss of a great deal of information in skeletal remains due to taphonomic processes makes this and even more classical medical definitions of health such as absence of disease unsuitable for paleopathological studies. Alternatively, biological anthropologists consider health from the perspective of physiological stress detectable in skeletal material (e.g., bones and teeth) as the most often preserved human tissues. Any adverse biological, physical, or chemical stimulus that disturbs homeostasis may produce stress (Tortora and Anagnostakos 1981). More precisely, in bioarchaeological context, stress is defined as "a measurable physiological disruption or perturbation that has consequences for individuals and populations" (Goodman and Martin 2002:12).

Assessment of health from skeletal remains includes determination of causative agents (if possible) and various other stimuli that influenced occurrence of stress markers. On a population scale, "it is a challenge to go beyond concerns with proximate causes of illness to examination of the broader and underlying social, economic, and political factors that determine pattern of nutrition, health, and mortality" (Goodman and Martin 2002:18). Goodman and Armelagos (1989) suggested the following model of stress:

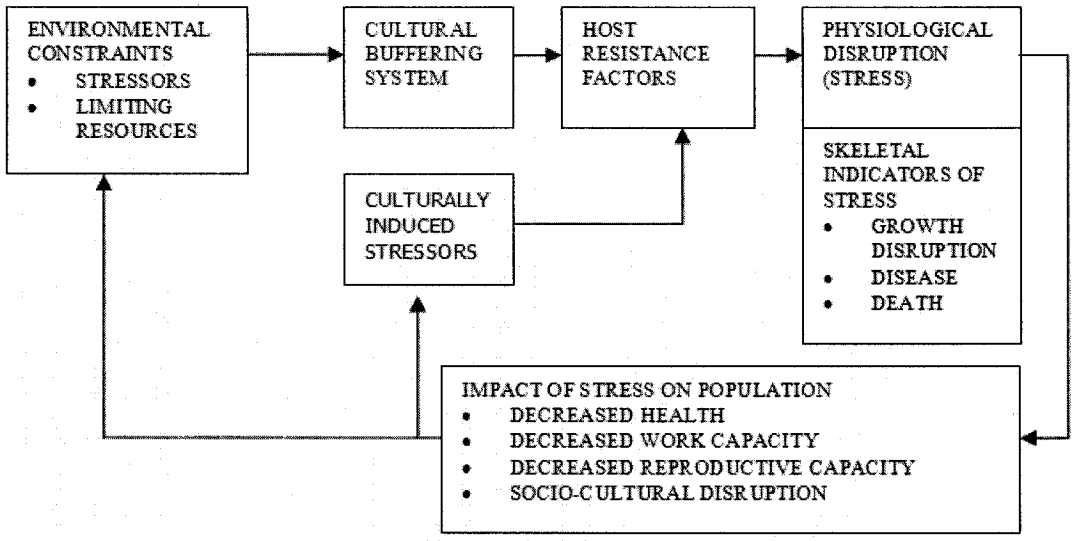

Figure 1.1. Stress model of physiological stress (adapted from Goodman and Armelagos 1989).

According to this model, multiple stressors, such as infectious diseases, traumas, and limited basic resources affected by climatic conditions comprise a set of environmental factors that influence an individual's health. Environmental factors can be buffered by the specific culture in place. For instance, invention of preventive vaccines and antibiotics created cultural barriers to serious infectious diseases that were the major cause of morbidity and mortality in the human past. On the other hand, culture may provoke or amplify environmental stresses. For example, aggregation of population in large settlements increased unsanitary conditions and favored the spread of infectious diseases (Sullivan 2005; Walker 1989).

The last buffer is host resistance. Personal characteristics or host factors, such as age, sex, genetic and immunologic predisposition or resistance, and personal behavior, play an important role in occurrence and severity of diseases (Gerstman 2003).

Evidently, a population often consists of a mix of individuals with different genetic predispositions and susceptibility to various diseases and different levels of immune competency strength. While genetic and immunity differences are difficult to trace in an archaeological context, host characteristics of age and sex provide important data on the pattern of stress distribution within and among populations. Thus, assessment of health in archaeological populations is based on the premise that if environmental factors are presumed to hold constant, the control for host variables allows the investigation of cultural factors that are associated with the variation in level of stress in a population (Goodman et al. 1984c).

From decades of such investigations, anthropologists have developed unique methodologies allowing reconstruction of the health of prehistoric populations based on stress markers in skeletal remains. In general, pathological bone formations observed in skeletal remains are subdivided based on specific versus non-specific etiology. Based on certain similarities in causes of stresses, Goodman and colleagues (1984c) suggest classifying stresses by three major categories: 1) general and cumulative stress, 2) general and episodic stress, and 3) specific disease stress. Commonly, paleopathologists are dealing with non-specific responses, which in this classification are identified as general. The non-specific character of many pathological lesions is related to the fact that bone tissue has a limited number of ways of reacting to various stresses activating osteoblasts (bone forming cells) or osteoclasts (bone destroying cells). It has been long recognized that different diseases can induce similar bone reaction. Despite the uncertainty

regarding etiology, non-specific lesions can serve as a measure of stress level in a population.

General stress markers are subdivided into those that accumulate over time and those that occur at a specific time. Studies of general and cumulative types of stress include analyses of mortality pattern and growth. Age at death is considered a strong indicator of stress (Acsadi and Nemeskeri 1970). The correlation between decreased age at death and other stress markers indicates the influences of stress on an individual's longevity (Bloom et al. 2005; Slaus 2000; Stodder 1997). Analysis of stress from the measurements of long bone length in subadults and adults is based on clinical research, which demonstrated that under stressful conditions the process of growth can be slowed down or stopped (Bogin 1988; Tanner 1978).

The most commonly observed episodic stress indicators are dental enamel hypoplasias and Harris lines. Hypoplastic lesions on dental enamel can result from the disruption in dental enamel formation due to various physiological stresses. Likewise, Harris lines, which can be observed on radiographic images, can form on long bones during their growth due to temporary disruption in the process due to physiological stress. One of the valuable characteristics of episodic indicators is that they allow an estimation of age at time of occurrence of adverse stresses.

And finally, there are many stresses that are considered specific. There are a limited number of specific pathologies, e.g., lesions that are produced by particular diseases, whose etiology can be clearly determined by differential diagnosis. Lesions related to some nutrient deficiencies (scurvy, iron deficiency anemia) and infectious

diseases (tuberculosis, syphilis) are examples of specific stresses. Ultimately, in the health assessment of a population, the use of several stress indicators allows a more comprehensive analysis of stress markers and their causative factors (Larsen 1997).

Important theoretical considerations in the health assessment of archaeological populations include the recognition of stresses that can leave marks on bone tissue versus stresses that are impossible to trace in skeletal remains and, consequently, the validity of inferring such stresses from the analysis of skeletal remains (Goodman and Martin 2002). Stresses can vary in duration, virulence, and pattern of how they affect the body. Multiple physiological stresses that cause various disorders and may lead to fatal outcome are restricted to soft tissue. Death or recovery from acute diseases, for example, often occurred fairly quickly, before infection could leave any pathological mark on the bone (Roberts and Manchester 1997). Normally, the process needs to be chronic and/or sufficiently severe to cause a bone reaction.

In this regard, Wood and colleagues (1992) speculated about the so-called osteological paradox. One point in their argument is that an absence of pathological scars on bone tissue is not an unambiguous indication of health. On the contrary, it is suggested that an individual without stress markers might not have sufficiently good health to overcome a stress. In turn, healed stress markers may sometimes indicate that an individual was fairly healthy and did not die but, instead, mitigated the disease(s).

Responding to these arguments, Steckel and Rose (2002) suggested that the "osteological paradox" can be tested by regression of the health index on demographic analysis of life expectancy. They hypothesized that if individuals with a higher

prevalence of stress markers had better health, they should have longer life spans than individuals who died without stress markers. On the other hand, if a prevalence of skeletal lesions reflects adverse impacts on an individual's health, then individuals with higher stress markers should (on average) demonstrate a shorter life span compared to individuals without stress lesions. Their data derived from 50 archaeological sites showed a significant positive correlation between a generalized health index based upon a variety of health indicators (including all those used in the present research) and life expectancy. Populations with higher health indices, e.g., with less stress indicators, demonstrated higher life expectancy. Thus, the prevalence of the skeletal lesions reflects a level of disease burden in a population (Goodman and Armelagos 1989; Larsen 1997).

Because data derived from a skeletal population depend on numerous factors, such as preservation of the material, mortuary practices that may bias the demographic picture, and other specific cultural and environmental factors, it should be analyzed and interpreted within an archaeological context (Buikstra and Cook 1980).

Archaeological Context

After many years of excavations and analyses of artifacts, the archaeological chronology for the San Francisco Bay area has been developed and a regional temporal framework for Central California has been refined. The primary division of temporal periods proposed herein was made by using grave lots as units for analysis of time-sensitive artifact assemblages supported by radiocarbon dates (Bennyhoff and Hughes

1987). There are three major temporal periods distinguished in the revised Central California Taxonomic System: Early, Middle, and Late (Bennyhoff and Hughes 1987).

According to Bennyhoff and Hughes (1987) Scheme B1, the Early period started during the Middle Holocene, approximately at 3000 BC, and continued to circa 500 BC. The authors distinguished the Early/Middle Transition period from approximately 500 BC to 200 BC followed by the Middle period, encompassing the time span up to circa AD 700. The period from AD 700 to AD 900 is the Middle/Late Transition period. And finally, the Late period continued from approximately AD 900 to the time of Spanish contact (AD 1769). Each major period is subdivided into phases based on changes observed in assemblage and artifact typology (e.g., beads, pendants, pipes, harpoons, etc.). In addition to divisions based upon temporal periods and phases, researchers distinguish three basic cultural patterns in Central California, highlighting changes in subsistence strategy, social organization, trade, settlement pattern, and mortuary and ceremonial practices. These divisions include the Windmiller Pattern (identified as "Lower Berkeley" for the San Francisco Bay area), the Berkeley Pattern, and Augustine Pattern (Bennyhoff and Fredrickson 1994).

The Early period is not considered in detail in this study because of the absence of archaeological strata representing this period in the examined site. In general, in addition to a wide distribution of milling stones used for processing hard seeds, the period is characterized by infrequent mortars and pestles, and relatively large numbers of projectile points, suggesting an emphasis on hunting (Bennyhoff and Fredrickson 1994; Moratto 1984).

The appearance of some sites of the Lower Berkeley Pattern in the San Francisco Bay Area is tied to the environmental development of marsh habitat, which provided a subsistence base for the local populations (Lightfoot 1997). The Middle period (Upper Berkeley pattern) in the San Francisco Bay area is marked by the intensive development of bay shore mounds. The increased number of sites dated to this period and the large number of individuals buried at these sites led researchers to hypothesize that there was a significant increase in population size and density during this time (Bennyhoff and Fredrickson 1994; Wilson 1999). This increase was possibly accompanied by a more sedentary style of living. Based on an elevated proportion of grinding implements relative to projectile points during the Berkeley Pattern as compared to the Windmiller Pattern, archaeologists have inferred a growing reliance upon an acorn economy (Bennyhoff and Fredrickson 1994:22). In the San Francisco Bay area, this period is characterized by an intensive tidal marsh economy (Hylkema 2002). The stress put on resource availability of a growing population and sedentary style of living may have contributed to an increased level of interpersonal violence throughout California (Andrushko et al. 2005; Lambert 1997). Analysis of mortuary complexes showed the development of a social hierarchy, seen in the concentration of grave goods among a few burials. Luby's (1991) cluster analysis of mortuary complexes showed slight differences in wealth and social position, suggesting that a non-egalitarian social system was already in place during the Middle period. The inferred social complexity, however, was not yet elaborate. Burials usually contained a limited set of accompanying items. Rarely, graves included a so-called "shaman's kit," e.g., quartz crystals, charmstones, bone whistles, or

animal bones, indicating ceremonialism represented mostly as shamanism (Bennyhoff and Fredrickson 1994:23).

In the Late period (Augustine Pattern) social stratification reached a more marked development. The sites dated to this period are represented by a rich assemblage of artifacts. Wealth and social status, expressed in terms of burial goods and type of funerary rite (e.g., inhumation vs. cremation or pre-interment grave pit burning), were unequally distributed among the populations (Lightfoot and Luby 2002). Cluster analysis of a mortuary complex from CA-SCL-38 indicated the development of a hierarchical society with age, gender, wealth, and social status differentiation (Bellifemine 1997). As evident from the abundance of mortars and pestles, the acorn increasingly became a staple of the economic base for the prehistoric populations of Central California. Increased numbers of recovered projectile points from this period suggests an increased emphasis on hunting as compared to the Berkeley Pattern (Hylkema 2002). A new technology incorporating the bow and arrow was introduced in the region in the Late period. Trade, indicated by the growing number of exotic goods that came from a relatively great distance, greatly increased during this period (Bennyhoff and Fredrickson 1994). Another hallmark of the Late period is a wide-spread geographic distribution of *Haliotis* banjo pendants, which are assumed to represent the ethnographically described Kuksu religion (Gifford 1947; Leventhal 1993).

The archaeological data from the region demonstrate a progressive development over time, which achieved cultural and economic florescence in the Late prehistoric period. At the same time, studies of faunal remains suggest resource depression in the

Late Holocene observed through evidences of a broad-spectrum diet, more intensive use of high-cost, low-rank prey (such as small mammals) versus low-cost, high-rank prey (such as large mammals), and exploitation of more distant vicinities to obtain food (Broughton 1994, 1999; Simons 1992). Simons (1992) analyzed vertebrate remains from several sites from the San Francisco Bay area and found a trend of significant decline in artiodactyls relative to increase in sea otters in the Late period. Broughton's (1999) study of faunal remains from the Emeryville mound (East San Francisco Bay) showed greater exploitation of distant patches and an increase in the proportion of more costly fauna over time.

Observed evidences of dietary changes are sometimes interpreted in the context of the resource intensification model, which was originally based on the Malthusian theory of population pressure (Broughton 1999). Population pressure is viewed as a trigger that induces changes in subsistence pattern and social structure (Boserup 1965). To overcome imbalance between the growing population and its mode of subsistence, people devote more energy to obtain necessary resources; i.e., they intensify their subsistence strategy (Broughton 1994). That is why the increase in number of faunal remains from small prey observed in the Late Holocene is viewed as evidence of resource depression. Increased incorporation of high-labor acorn technology in a group's subsistence base is also considered as a resource intensification strategy adopted in response to stress from population/resource imbalance (Basgall 1987).

Various causative factors are considered for the social and economic development of the prehistoric societies in the Late Holocene. Basgall suggested that "requisites of an

acorn-based economy ... appear to be behind the organizational complexity evident in much of late prehistoric and protohistoric California" (1987:45). In contrast, Hildebrandt and McGuire (2002) argued that subsistence activities, hunting in particular, should not be viewed only in terms of a provision supply, but should be tied to various symbolic aspects of culture, kinship pattern, and political authority as well. Other authors, emphasizing the role of social factors, link the emergence of some social traits to environmental influences (Arnold 1992, 1995; White 1998). A period of environmental instability, or the so-called Medieval Climatic anomaly (approximately AD 800–1350), which is characterized by periodic droughts and warmer seawater, has been detected in many places within California (Ingram et al. 1996; Jones et al. 1999; Stine 1994). In the San Francisco Bay area the effect of warmer climatic conditions was identified through evidence of reduced freshwater inflow to the bay from the San Joaquin Delta, which likely resulted from reduced precipitation (Byrne et al. 2001). Lightfoot and Luby (2002) linked the period of climatic instability with the abandonment of several sites in the San Francisco Bay region. Arnold (1992) suggested that the development of cultural complexity can be linked to the economic control over bead manufacture, microlith industry, and trade, which an emerging elite exercised under unstable environmental conditions.

Theoretical models built on archaeological data provide different interpretations and predictions for health consequences related to observed increased sedentism, population density, social stratification, and shift in subsistence pattern (e.g., an intensive exploitation of acorn technology, a broadened diet at the expense of low-rate prey, or a

focus on marine resources in some regions). Resource intensification theory predicts that decline in foraging efficiency would lead to an increase in physiological stress and a greater risk of malnutrition (Broughton and O'Connell 1999). The theory also argues that a growing reliance on certain plant resources as a staple (such as the acorn for Central California) would lead to malnutrition and a subsequent increase in morbidity and mortality (Cohen 1977).

Alternative views consider such a shift of subsistence base to greater consumption of highly productive resources, when measured in terms of calories per land area as well as storable food resources, as a successful cultural adaptation. Reliance on storable food is seen as a buffer from stresses related to food shortage during lean seasons and, accordingly, beneficial for human survivorship and health (cf. Roosevelt 1984).

Anthropological Studies from California

There is a series of bioarchaeological studies that investigated the consequences for health of California prehistoric populations of multiple social and economic changes that were observed archaeologically. Many of the studies demonstrated health decline over time (Ivanhoe 1995; Ivanhoe and Chu 1996; Lambert 1993; Walker 1986; Walker and Lambert 1989, 1991; Weiss 2002), while others presented controversial results of improvement of one health variable along with deterioration of the others (Dickel et al. 1984; Doran 1980; McHenry and Schulz 1978).

The authors who observed deterioration of health over time correlated their findings with dietary insufficiency and an elevated load of infectious diseases; both of these factors were presumably related to increase in population size and density and were further exacerbated by environmental instability during the global warming period. Walker (1986) and Lambert and Walker (1991) analyzed cribra orbitalia, i.e., porosity of the eye orbit attributed to iron-deficiency, among the hunter-gatherer populations from the Santa Barbara Channel region. The authors found that frequency of lesions increased at the end of the Middle period. Walker (1986) and Lambert and Walker (1991) attributed increase of cribra orbitalia to bacterial infections obtained through the sources of drinking water, which might have been contaminated because of their scarcity during dry climatic conditions and consequent heavy use by people and animals. An archaeologically observed shift of subsistence base toward increased reliance on marine food also might have indirectly influenced the prevalence of orbital lesions caused by the consumption of raw fish and mammals infested with helminthes parasites (Lambert and Walker 1991; Walker 1986).

Lambert (1993) studied the health consequences of the shift in subsistence base from the broad spectrum maritime hunting-gathering diet to one with heavy reliance on fishing for the Santa Barbara prehistoric populations. The author emphasized that a diet rich in protein and other necessary elements did not protect the population from an increased frequency of periostitis (marker of infectious diseases) and a decline in stature, indicating deterioration in health at the end of the Middle period. Lambert (1993) attributed decline in health to increased population size, density, and intensification of

trade, which influenced the spread and, possibly, caused the introduction of new infectious diseases among the studied populations. The author also suggested that the warmer seawater and drought evidenced during the Middle period might have diminished marine and terrestrial food and drinking water resources, creating stressful conditions for population health.

Several studies from Central California also observed negative health changes in prehistoric populations over time. In Central California, evidence of malnutrition was related to the increased use of the acorn as a staple (Ivanhoe 1995; Ivanhoe and Chu 1996) and to environmental stresses during the global warming period (Weiss 2002). Ivanhoe (1995) and Ivanhoe and Chu (1996) studied changes in cranioskeletal size in prehistoric populations from the Central Valley and the San Francisco Bay area. Comparing the osteometric data of 359 individuals from three temporal horizons (Early, Middle, and Late), Ivanhoe (1995) concluded that a secular trend of decrease in cranioskeletal size observed in the Central Valley correlates with a shift in subsistence base and an increase in demographic stress for females. In this view, a shift from a broad spectrum hunting economy toward a greater reliance on acorns and a higher birth rate led to a calcium deficit and subsequent stunting. The study of the San Francisco Bay area sample also revealed a decline in craniofacial size over time, but the results were not as linear as in the Central Valley and, for the most part, reached only marginal statistical significance (Ivanhoe and Chu 1996). The authors found that the cranioskeletal parameters dropped dramatically between the Early and beginning of the Middle periods,

remained relatively stable during the Middle and Phase I Late periods, and then decreased slightly in the Phase II Late period.

Weiss (2002) compared femoral cortical thickness of prehistoric populations from two San Joaquin Valley cemeteries, one of which (cemetery I) predated the drought at 2895 ±160 yr BP to 1845 ± 90 yr BP and the other of which (cemetery II) was used at the time of drought and dated from 1100 ± 90 yr BP to 1220 ± 200 yr BP. It was found that cortical thickness decreased in the population from cemetery II along with the age at death, especially for females, while pathology/trauma indices increased. Because both populations demonstrated cultural similarity, relatedness, and had acorn as a staple of their diet, the author proposed environmental factors related to prolonged periods of drought were causative factors of health decline in the population from cemetery II.

Several other studies present a complex picture of stress distribution over time and space in California. Ruff (1975) found that frequencies of Harris lines (markers of growth arrest associated with physiological stress) showed similar rates in the samples from the Early and Late periods from the San Francisco Bay area. At the same time, McHenry and Schultz' study (1978), based on a sample from Sacramento-San Joaquin Valley, observed decline in frequency of Harris lines from the Early to Late period. While prevalence in Harris lines is associated with seasonal stress and related to scarce food resources during the winter and early spring period, their decrease in later temporal periods is attributed to increased reliability of the stored food. This study also found that prevalence of enamel hypoplasia (marker of physiological stress on dental enamel) decreased from the Early to Middle period, but then increased again in the Late period,

indicating increased morbidity in the Late period. Likewise, Doran's demographic analysis of the subsamples from three temporal periods also presents complex results of decreasing mortality of subadults after age 4 but declining adult survivorship over time (Doran 1980). Dickel and colleagues summarized the results of several health studies from the lower Central Valley and suggested that "nutritional specialization in storable food resources was not accompanied by a uniform increase in good health, but possibly represented a trade-off of periodic acute stress for chronic stress" (1984:450).

Bartelink's (2006) study of diet and health trends in the Sacramento Valley and the San Francisco Bay area presented a complex picture with significant variation between the regions. While the analysis of stable isotopes for the Sacramento area indicated no change in proportion of dietary protein and carbohydrates, the results from the San Francisco Bay sites showed a clear shift in subsistence pattern from reliance on large marine mammals toward greater consumption of terrestrial resources such as shellfish, terrestrial mammals, and C3 plants, demonstrating consistence with resource intensification theory. On the contrary, assessment of health in both regions revealed that the Sacramento area showed more correlation with the intensification model indicating a trend of health deterioration from the Early to Late period. No significant changes over time were observed in the San Francisco Bay area except for a decreased prevalence of enamel hypoplasias (dental marker of physiological stress), noted in one of the tooth types. The latter finding allowed the author to suggest improvement of health in the San Francisco Bay region in the Late period.

Research Questions and Hypothesis

The goal of the present study is to investigate whether environmental and cultural changes affected the health of the prehistoric population from the CA-Ala-329 site. Several indicators of physiological stress, such as dental enamel hypoplasia, porotic hyperostosis, osseous signs of infectious diseases, and stature are examined, and mean age at death is calculated.

Based on the information derived from archaeological data presented above, a research hypothesis was developed.

Research hypothesis. It is hypothesized that the health of the prehistoric population from CA-Ala-329 site declined with time due to an increase in population density in the region and possible consequent resource depression. If this is correct, the prevalence of selected skeletal markers of enamel hypoplasia, porotic hyperostosis, and infectious diseases should increase in the Late period, while stature should decrease.

In parallel to testing this hypothesis, several research questions are investigated in this study:

1. Did the economic, cultural, and environmental changes differentially affect males and females of the examined populations?
2. Did the pattern of stress distribution between adult and subadult segments of the population change over time?
3. Was an individual's longevity affected by compromised health presumably correlated with the observed stress markers?

Sample Description

The skeletal sample used in this study comes from the Ryan Mound, CA-ALA-329 archaeological site, whose inhabitants belonged to the ethnographic East Bay Ohlone (Costanoan) speaking tribal groups. CA-ALA-329 is located on the southeastern side of the San Francisco Bay in the Coyote Hills area. Once situated on the edge of the Bay Shore zone, the area regularly flooded during rainy seasons, the mound now lies approximately two and a half miles inland after the construction of the levee (Coberly 1973). The territory along the east side of the bay is characterized as rich in various food resources provided by bayshore, salt and fresh water marsh communities, and adjacent grasslands (Leventhal 1993).

The temporal framework for the Ryan Mouond was determined based on a series of 14 radiocarbon dates derived from human collagen samples and 21 shell beads, 116 obsidian hydration measurements, the analysis of *Olivella* shell bead types following the Bennyhoff and Hughes (1987) Scheme B1, and the stratigraphic distribution of the burials within the mound (Leventhal 1993). The results from these analyses span three temporal periods: Late Middle period (150 BC–AD 900), Phase I Late period (AD 900–AD 1500), and Phase II Late period (AD 1500–AD 1700) (Note: Phase I Late period and Phase II Late period will be referred in this work as Late I and Late II periods respectively).

The northern portion of the site was partially excavated by San Jose State University between 1962 and 1968. The skeletal population curated at San Jose State University comprises 285 field-assigned burial numbers, which represent approximately 320 individuals. Jurmain (1990) performed detailed osteological analysis with determinations of age and sex based on established criteria. The demographic distribution of the sample is presented in Table 1.1. Subsamples for each stress marker examined in this study are presented separately in the Results sections for each stress variable.

Table 1.1. Demographic Distribution of the CA-Ala-329 Sample

Time Period	Males	Females	Adults of Undetermined Sex	Adults of Undetermined Age	Total Adults	Subadults (0-15)	Total
Middle	18	16	3	2	39	19	58
Late I	39	47	7	17	110	47	157
Late II	34	25	3	18	80	25	105
Total	91	88	13	37	229	91	320

CHAPTER 2: LITERATURE REVIEW FOR USED VARIABLES

Enamel Hypoplasia

Enamel hypoplasia is a decrease in enamel thickness caused by temporary disruption in the dental enamel forming process (Goodman et al. 1985). Enamel formation commences during the intrauterine stage of development for the deciduous teeth and ends at about puberty for permanent teeth (Skinner and Goodman 1992). Since tooth enamel does not undergo remodeling after being formed, hypoplastic defects provide us with an accurate record of childhood stresses.

Enamel defects occur in a variety of types. They may appear as lines or furrows that encircle the tooth, pit, array of pits or circumscribed area of thinner enamel localized only on the labial surface and may affect one tooth or many, be symmetrical or asymmetrical. Federation Dentaire International (FDI) standardized the recording system and classified the ranges of appearances of enamel hypoplasias as pits (type 3), horizontal grooves (type 4), vertical grooves (type 5), and missing enamel (type 6) (FDI 1982, 1992). Diversity of appearance of the enamel defects was taken into consideration in this classification system. Thus, for example, type 4, may be presented by horizontal lines, grooves or furrows. Hypoplastic defects of type 4 are the most common type of enamel hypoplasia. They are often referred to as linear enamel hypoplasia (LEH). Type 3 defects occur much more rarely and method of evaluation of these defects is undetermined (Goodman and Rose 1990). Defects of type 5, vertical grooves, are

typically not considered in studies of physiological stresses in archaeological populations because the etiology of these defects is not understood. Type 6 defects with areas of missing enamel and exposed dentin are the severest expression of hypoplasia.

Two types of enamel hypoplasia are typically observed in deciduous teeth: linear enamel hypoplasia and localized hypoplasia of primary canine. Linear enamel hypoplasia in the deciduous dentition may be represented by lines, grooves, or an array of pits. Localized hypoplasia of the canine is a specific enamel defect that is characteristic only of the primary dentition. It could be circular or ovoid in shape and restricted to labial surface of the deciduous canine (Guatelli-Steinberg 1999).

Biomechanism of Enamel Formation and Enamel Defects

Enamel Formation. Enamel formation is called amelogenesis. This process includes two major parts: secretion of enamel matrix and calcification of the enamel. Enamel formation begins when ameloblasts separate from the rest of mesenschimal epithelium and, together with odontablasts, start to create a tooth germ. The ameloblastic layer starts the secretion and apposition of the enamel matrix at the line that will become the dento-enamel junction at the occlusal surface of the future tooth. Enamel matrix is structured into enamel prisms (rods) of characteristic keyhole shape (Skinner and Goodman 1992). The appositional process continues to lay down a new layer of enamel over the old one, forming tree-like rings (Massler 1941). Each transverse layer of the enamel built by microscopic enamel rods is laid concentrically from dento-enamel junction to the crown surface. These incremental layers, called striae of Retzius, can be seen under the microscope in a cut section of the enamel (Hillson 2002). Perikymata,

which is observed as a "staircase pattern" on the dental crown, is a surface manifestation of the striae of Retzius (Goodman and Rose 1990).

The next stage of the enamel formation after apposition of the enamel matrix is enamel calcification. The enamel matrix, which is made up of approximately 20% protein, 5–10 percent calcium salts, and water, begins to lose its water and protein during calcification (Nikiforuk et al. 1981). Besides primary calcification at the time of enamel apposition, the process of maturation includes three more consequent steps of increase in mineralization (Suga 1983). At the final stage, organic components and water are replaced by calcified crystals, which constitute 96% of the inorganic calcium-phosphate-carbonate component of the enamel (Nikiforuk et al. 1981).

Enamel Defects. Prolonged and severe physiological stresses that occur at the time of active amelogenesis negatively affect physiology and metabolism of ameloblasts and induce permanent alteration of enamel structure (Goodman and Rose 1990). Disruptions during the stage of enamel matrix secretion lead to formation of enamel hypoplasias. The size of the defects relates to the severity of stress (Suckling 1989). Enamel defects are expressed as linear decreases in thickness of enamel that follows the pattern of perikymata (Hillson 2002). Histologically, hypoplasia is marked by convergence of the striae of Retzius near the enamel surface, abnormal shape and configuration of the prism at the site of the defect (Skinner and Goodman 1992). To distinguish from normal striae of Retzius, pathological striae are referred to as Wilson bands. In approximately half of the cases, Wilson bands mark the beginning of the

hypoplasias, while in the other half, pathological striae take a random position within enamel defects (Goodman and Rose 1990).

Disturbances during the process of calcification lead to hypocalcification expressed as changes in color and opacity of the enamel. It was found, however, that hypocalcification may also occur due to disturbances during matrix secretion (Suckling 1989). However, the complex process of enamel maturation has not yet been studied sufficiently to know the duration of each stage of calcification. Consequently, it is impossible to determine the time of formation of this type of enamel defect. Thus, hypocalcification is not considered in the present study.

Etiology

Over 100 various causes can disrupt amelogenesis and lead to formation of hypoplastic defects (Cutress and Suckling 1982). In general, etiology of enamel hypoplasias can relate to three major factors: 1) hereditary disorder; 2) local trauma; and 3) systematic physiological stress (Goodman and Rose 1990; Suckling 1989). Three types are usually distinguished by their appearance. Defects due to hereditary abnormalities, which are referred to as amelogenesis imperfecta, present the most severe cases of hypoplasias. They involve all elements of both deciduous and permanent teeth, and are not attributed to a particular chronological period of enamel formation (Hillson 2002). These enamel defects constitute less than one percent in contemporary populations (Goodman and Rose 1990). Hypoplasias of localized traumatic and inflammatory origin affect one or several adjacent teeth, mostly maxillary central and lateral incisors (von Arx 1993). This type of hypoplasias is also relatively rare in modern

and prehistoric populations (Goodman et al. 1980; von Arx 1993). Both of these types do not relate to physiological disturbances due to environmental stresses and are not considered in this study.

Hypoplasia due to systematic stress is the most common type of hypoplastic defects. This type is recognized by the linear nature of enamel defects that follow the pattern of perykimata, reflecting specific time of systematic stresses, and by chronological match of enamel defects between teeth formed during the same time of enamel development (Goodman et al. 1984; Hillson 2002). A variety of other clinical conditions have been related to occurrence of this type of hypoplasia. Strong correlation was noted between enamel defects and infectious diseases, such as rubella, tetanus, and syphilis (Pindborg 1982). Other studies have shown an association of enamel hypoplasia and such peri- and neonatal causes as maternal diabetes (Noren 1984), birth at less than 32 weeks of gestational age with signs of asphyxia, low birth weight children with disturbances of vitamin D metabolism, and cerebral palsy (Brook 1997). It was also demonstrated that high concentrations of fluoride in the drinking water in severe cases leads to enamel pitting (Driscoll et al. 1986). A high percentage of enamel defects was observed in children with mental retardation, hearing defects, and various other neurological disorders, with heightened prevalence in cases with a history of bacterial diseases (Bhat 1989; Martinez et al. 2002). The majority of etiological factors, however, are rare conditions that do not explain the high frequencies of enamel defects reported in many populations (Guatelli-Steinberg and Lukas 1999). Experimental studies on animals and anthropological studies among living populations demonstrate a strong association of

enamel hypoplasia with physiological stresses related to infectious diseases and malnutrition.

Influential Factors

Role of Infectious Diseases. Many studies linked occurrence of enamel hypoplasia with infectious diseases. May and colleagues stated that "illness is but one component contributing to the attainment of a threshold level of physiological stress necessary to impair ameloblastic function" (1993:46). Sarnat and Schour (1942) successfully matched 29 out of 60 cases of hypoplastic defects with diseases known from medical histories of the patients. The medical records included such diseases as chickenpox, diarrhea, diphtheria, measles, pneumonia, scarlet fever, and whooping cough. Hillson and his colleague (1992), using a more precise methodology of recording hypoplasia, were able to match medical records and enamel defects in 70% of the cases.

Some studies demonstrated an association between hypoplastic defects and particular infectious diseases in individuals with known medical history. For instance, Suckling and co-workers (1987) in their study of over 1,000 New Zealand children found that enamel defects prevailed in those individuals who had chickenpox before age 3. A study of 141 Danish children revealed that in 22.7% of cases, enamel hypoplasia was associated with gastrointestinal disorders accompanied by non-specific diarrhea (cf. El-Najjar et al. 1978).

Experimental studies with animals also confirmed association of enamel hypoplasia and infectious diseases. For instance, a series of studies performed by Kreshover (1960) in the middle of the 20th Century demonstrated formation of enamel

defects in rats infected with pathogenic viruses and bacteria. Suckling and colleagues (1983) showed formation of hypoplastic defects in sheep due to parasitic infestation.

Role of Nutrition. Many studies show a high correlation of enamel defects and malnutrition (Enwonwu 1973; May et al. 1993; Sweeney 1971; Zhou and Corruccini 1998). Studies on this topic were conducted among various contemporary populations living in adverse conditions. For instance, Enwonwu (1973) studied Nigerian children from different socio-economic strata. He found that children from the high socio-economic group had no hypoplasia. In contrast, the frequency of enamel defects in children from the low socio-economic group was 21%. Malnutrition was considered the major factor in prevalence of hypoplastic defects among underprivileged Nigerian children. Goodman and colleagues (1987) have recorded one or more hypoplastic defects in 46.7% of 300 children from rural Mexico that have been considered as a mildly to moderately undernourished population. In contrast, industrial countries have much lower prevalence of enamel hypoplasia. The rate of hypoplasia in English children recorded by Brook and co-workers (1997) was 14.6%, while Cutress and Pierce (1984) have found less than 10% of hypoplastic defects in New Zealand children.

Studies focusing on supplementation programs present vivid evidence of influence of malnutrition on formation of hypoplastic defects (Goodman et al. 1991; May et al. 1993). Goodman and colleagues (1991) studied the influence of nutritional supplementation on prevalence of dental hypoplasia using a sample of 84 Mexican children from a rural Aztecan community. The authors found that children who did not

receive supplemental food had two times more hypoplastic defects (74.4%) than children in the supplemented group (39.5%). The control group that did not receive additional nutrition had a greater proportion of dental defects formed before age 1.5 and after age 3.

Zhou and Corruccini's study (1998) of the prevalence of enamel hypoplastic defects considered the impact of a documented historical famine in China (1959–1961). This crisis caused the death of about 30 million people in three years. The results of this study presented even more convincing evidence of a high correlation between dental hypoplastic defects and nutritional stress. The study, based on a sample of 3,014 individuals, demonstrated significant prevalence of the hypoplasias among the individuals whose teeth were developing during the time of famine (60.6%) comparing to the post-famine group (42.2%). The investigators recorded the highest frequency of hypoplasia in rural areas that had the most adverse nutritional and living conditions.

Generally, it is difficult to distinguish dietary deficiency of particular nutrients because malnutrition in human populations is usually characterized by insufficient intake of multiple nutrients. Experimental studies on animals, however, were able to examine the relationship between specific nutrient deficiencies and enamel defects (Diorio et al. 1973; Grahnen and Selander 1954; Mellanby 1934).

Synergism of Factors. Because of multifactorial etiology, it is difficult to determine the exact cause of enamel defects. Numerous anthropological studies, however, demonstrated that nutritional deficiency, infectious diseases, and synergetic interaction of both are the major etiological factors of enamel hypoplasia in

disadvantaged populations (Goodman et al. 1991, May et al. 1993). Interaction between malnutrition and infectious diseases is complex. On the one hand, malnutrition negatively affects response of the immune system to diseases predisposing an organism to infections; on the other hand, severe, prolonged and repeated infections associated with insufficient nutrient intake, depletion of necessary nutrients, fever, and impaired metabolism influence nutritional status (King and Ulijaszek 1999). Thus, since enamel hypoplasia can be related to numerous causes, it is viewed as a non-specific marker of physiological stress (Goodman and Rose 1990).

Anthropological studies in developing countries emphasize synergism of malnutrition and infectious diseases as the key etiological consideration. May and colleagues's (1993), for instance, performed a study of the prevalence of enamel hypoplasia in relation to nutritional supplementation and morbidity among malnourished Guatemalan children. The results showed that children who received less than 34.25 kcal/day had more enamel hypoplasia than those who received more supplemental food. At the same time, hypoplastic defects were more prevalent in children who were ill more than 3.6% of the time. Goodman and co-workers (1991) in their study of malnourished Mexican children also found greater frequency of hypoplasia in malnourished children. Higher prevalence of enamel defects in females from this sample was associated with increased morbidity.

Studies on Archaeological Populations

Causative Factors. Many bioarchaeological studies examined changes in prevalence of enamel hypoplasia with changes in subsistence pattern. One of the major

foci relating to this topic concentrated on the investigation of changes in physiological stress during the transition from hunter-gatherer to agricultural subsistence strategy. In these studies, an increase in frequencies of enamel defects with the adoption of agriculture suggested that malnutrition, due to a narrow spectrum of diet and strong reliance on one carbohydrate-type of food as a staple, was one of the major etiological factors (Cucina 2002; Goodman et al. 1980; Larsen 1984; Pechenkina et al. 2002). For instance, Goodman and colleagues' study (1980) examined prevalence of enamel hypoplasia in skeletal samples of prehistoric North American populations (Illinois) from three temporal periods. The frequency of occurrences of enamel hypoplasia was compared between the Late Woodland period, when subsistence economy was based on a wide range of local flora and fauna, the Mississippian Acculturated Late Woodland that is characterized by introduction to maize cultivation, and the Middle Mississippian period characterized by increased population density and a subsistence pattern based on maize. It was found that enamel hypoplasia gradually increased from 45% in Late Woodland to 60% in Mississippian Acculturated and to 80% in the Middle Mississippian period. Higher incidences of enamel defects in later periods are explained by the change of subsistent pattern to one with heavy reliance on maize agriculture.

As in clinical research among living groups, many osteologists suggest synergetic interplay of malnutrition and infections as an etiological explanation of enamel hypoplasia (Littleton 2005; Stodder 1997; Walker and Lambert 1989). Walker and Lambert (1989) compared the prevalence of hypoplastic defects in a sample from one of the Santa Barbara sites dated to AD 100–1100 with the prevalence of hypoplasias

recorded for other sites in this region that both predated and postdated this site. The target population had almost twice as high a frequency of enamel defects than did populations from the other temporal periods. The authors emphasized the coincidence of site occupation with a period of climatic instability. Stressful environmental conditions induced a decrease in food and drinkable water resources. In the authors' opinion, food shortage and spread of infectious diseases, further complicated by an increase in local population density, were responsible for the prevalence of hypoplasia in the studied population.

Correlation with Longevity. Some authors have discovered an association between increased frequency of hypoplastic defects and decreased longevity (Goodman and Armelagos 1988; Palubeckaite et al. 2002; Slaus 2000; Stodder 1997). This pattern has been also found in a Plio-Pleistocene sample of fossil hominids (White 1978). It is suggested that childhood stresses expressed in enamel defects bring "biological damage" to individuals weakening their ability to respond to other stresses (Goodman and Armelagos 1988). For example, Palubeckaite and co-workers (2002) examined prevalence of hypoplastic defects in three samples from Medieval Danish and Lithuanian populations. One sample from a Lithuanian Medieval town that had the highest frequency of hypoplasia exhibited a correlation between more prevalent and severe hypoplasia (three or more stress episodes per individual) and shorter life span (mean age at death <30). Other authors failed to find a relationship between prevalence of hypoplasia and shortened life span (Saunders and Keenleyside 1999).

Sex Differences. Numerous studies investigated sex differences in prevalence of physiological stress expressed in hypoplastic defects. Males have commonly been reported to have higher occurrence of enamel hypoplasia than females, with the explanation assuming they are less buffered than females against environmental stresses during childhood development (Palubeckaite et al. 2002; Saunders and Keenleyside 1999; Van Gerven et al. 1990). Saunders and Keenleyside (1999) examined a historic sample of 253 adults from Canada for prevalence of enamel hypoplasia. All individuals, according to historical descriptions, were from middle to high socioeconomic strata. Analysis of maxillary and mandibular canines demonstrated significantly higher frequency of enamel hypoplasia in males than in females. Historical records reported that undernutrition was not a factor, even in poor strata of this community. On the other hand, various diseases and, most notably, epidemics of infectious diseases were very common. Thus, the authors attributed prevalence of hypoplasia in males to their higher susceptibility to physiological stresses during childhood.

Results of the other studies, however, demonstrated that sex differences might not be that easily detectable and interpreted. Slaus (2000), for instance, examined prevalence of hypoplasia in a 19th Century Croatian sample and found significantly greater frequency of hypoplasia in females (48.2%) than in males (9.7%). Ethnographic records from the region do not show evidence of preferential treatment of males over females. Still, the author suggested different social status and, thus, access to food as one of the possible but not demonstrated explanations. Other investigators also have not found sex differences

in prevalence of enamel hypoplasia (Goodman et al. 1980; Lanphear 1990; Malville 1997).

Summary

Enamel formation commences during the intrauterine stage of development for deciduous teeth and ends at about puberty for permanent teeth. Disruptions during amelogenesis induce formation of hypoplastic defects. Enamel hypoplasias do not undergo remodeling later in life, serving as a permanent record of childhood physiological stresses. Etiologically, enamel hypoplasia relates to multiple causes and, thus, it is viewed as a marker of non-specific physiological stress. Many anthropological and clinical studies demonstrated that malnutrition and various infections are main factors that influence occurrence of hypoplasia. Because of complex interaction between malnutrition and infectious diseases, anthropologists emphasize synergism of both factors as the best etiological explanation for development of enamel hypoplasias.

Porotic Hyperostosis

Porotic hyperostosis is defined as pathological change in bones that is seen macroscopically as pitting or spongy-like lesions (Goodman and Martin 2002). Porotic lesions have been assumed by most paleopathologists to be associated with anemia (Goodman and Martin 2002; Palkovich 1987; Stuart-Macadam 1985). Recent studies, however, demonstrated that careful differential diagnosis is crucial to distinguish porotic lesions due to possible anemia from other causative conditions, such as inflammation,

osteoporosis (Walper et al. 2004), and scurvy (Brickley and Ives 2006; Ortner et al. 1999).

Because of the general response of the body to anemia, skeletal changes of metaphyseal and cranial bone remodeling (i.e., coarsening of trabeculae and cortical thinning) are common characteristics of all anemias, except sickle cell disease, which is distinguished by bone infarcts (Aufderheide and Rodriguez-Martin 1998). It is necessary to consider geographical and ethnic affiliation of the sample, severity of pathological expressions, and anatomical patterning of pathological involvement to differentiate anemias. In nutritionally derived anemia porotic lesions are usually distributed symmetrically on the ectocranial surface of the frontal, the superior surface of the orbits (cribra orbitalia), parietals, and less frequently on the occipital bone (Buikstra and Ubelaker 1994). In severe hereditary anemias porotic changes also affect tubular bones (Aufderheide and Rodriguez-Martin 1998). In the early stages, porotic hyperostosis is represented only by fine, scattered foramina, while in more fully developed cases, affected areas are remodeled by the enlarged diploe and the outer layer of compact bone becomes thinner or disappears completely (Goodman and Martin 2002). Radiography demonstrates changes of trabecular organization to a more coarse texture with the characteristic "hair-on-end" pattern (Stuart-Macadam 1992).

Biomechanism of Porotic Hyperostosis

Porotic lesions result from an increase in erythropoiesis—i.e, the production of red blood cells (Hoffbrand and Pettit 1993). Trabecular bone is found in vertebrae, flat

bones (diplöe of the cranium), and in long bones (marrow), and it is within this type of bone that hemopoietic tissue produces red blood cells (erythrocytes). During the active period of growth in utero and in infancy, the red blood cells are produced also outside the bone marrow in the spleen, liver, and adrenal cortex (Aufderheide and Rodriguez-Martin 1998). The major physiological component of red blood cells is hemoglobin, which contains two types of globin chains and a heme molecule that contains iron. Through the action of hemoglobin erythrocytes' main function is to bring oxygen from the lung to the tissue cells, whose metabolism depends on oxygen supply. The oxygen is transported by binding to the iron molecule of hemoglobin. Inadequate amount of dietary iron and essential vitamins, abnormality in either type of globin chain, or genetically induced inadequacy of hemoglobin production leads to subnormal level of circulating hemoglobin. A level of hemoglobin lower than 13.5 g/dl in males and 12.0 g/dl in females stimulates the production of more red blood cells in marrow (Aufderheide and Rodriguez-Martin 1998). Intense pressure created by the proliferating marrow cells produces remodeling of the trabeculae and resorption of compact bone (Trueta 1968).

Etiology

By definition, all types of anemia are characterized by a decrease in the total red blood cell mass (Uthman 1998). Different pathological expressions, causes, and complications of this clinical condition result in a variety of anemias. Anemia varieties of inherited and acquired types are subdivided based on the mechanism of the disorders into disorder of heme synthesis (such as iron deficiency and anemia of chronic diseases), bone marrow failure (aplastic anemia), biomechanical lesion in DNA synthesis

(megaloblastic anemia), defects of red blood cell metabolism or injuries at the red cell membrane (hemolytic anemias), and defects in hemoglobin and hemoglobin synthesis (sickle cell anemia, thalassemia, and other hemoglobinopathies) (Rifkind et al. 1986). Most kinds of anemia are rarely occurring disorders. Frequencies of anemia higher than 1% in living populations are attributed to hereditary anemias or acquired anemia due to malnutrition and infections, which are the most common types of anemia in the world (Callender 1985; Klepinger 1992; Uthman 1998).

Hereditary varieties of anemia are inherited disorders at a genetic level that lead to decreased synthesis of a globin chain (thalassemia) or production of a globin chain with altered structure (hemoglobin S, C, and E anemias, and some other hemoglobinopathies) (Rifkind et al. 1986). Inherited anemias are a characteristic feature of particular regions or populations. Hemoglobin S anemia is the most notable and widespread type of the hemoglobinopathies, which in the form of sickle cell disease is accompanied by pain and tissue necrosis (Shin and Bellenir 1998). The place of origin of hemoglobin S disease is usually considered to be in the Mediterranean and especially in Africa, where the percentage of gene carriers is as high as 45% (Uthman 1998). Hemoglobin E, the second most common anemia in the world, is a mild anemia that is found in Southeastern Asia, where it prevalence constitute 10–20% of the population (Uthman 1998). Thalassemia of various clinical severities, from clinically undetected to incompatible with life alpha-thalassemia, and debilitating beta-thal major, is confined to tropical regions, except American continents. The vast majority of people with inherited anemia are gene carriers or have a mild form of anemia, such as hemoglobin E and

thalassemia minor, which in clinical practice could without proper laboratory tests be misdiagnosed as iron deficiency anemia (Uthman 1998).

The most common types of anemia in the world occur due to lack of the building materials necessary for normal function of red blood cells, such as iron (leads to iron deficiency anemia), vitamin B12 and folic acid deficiency (megaloblastic anemia). According to the World Health Organization study from the 1980s, 18% of males, 35% of all females, and 51% of pregnant women in the world were diagnosed with nutrient deficiency anemia (Fomon and Zlotkin 1992). The worldwide problem exists mostly due to poor availability of necessary forms of nutrients at the time of increased internal demands and excessive blood loss (Rifkind et al. 1986). Another common type of anemia is anemia of chronic disease, which occurs due to a natural defensive reaction of the human body to infectious diseases (Weinberg 1992). In clinical practice it is found mostly in mild forms (Rifkind et al. 1986). Despite the frequency of occurrence, this type of anemia is the least understood (Uthman 1998).

Iron Deficiency Anemia

Biomechanism. In the human body, iron is an essential element, and its balance is precisely regulated. It is present in hemoglobin (red blood cells), myoglobin (muscles), enzymes (various tissues), transferrin (plasma), ferritin and hemosiderin (macrophages of the reticuloendothelial system) that serve in the transport, utilization, and storage of oxygen (Rifkind et al. 1986). The greatest content of iron (80%) is in the heme component of hemoglobin within red blood cells (Dallman et al. 1980). The body normally regulates the quantity of iron within narrow limits to maintain a relatively fixed

amount. A distinctive feature of iron metabolism is recycling of iron. At the end of the life span of a red blood cell (100–120 days), iron is removed by the spleen and sent back to marrow for reuse in new hemoglobin. Accordingly, the daily amount of iron lost every day is usually very small, around 1 mg in a normal male (Callender 1985).

To maintain homeostasis, the replenishment of iron is accomplished through dietary intake. The amount of iron absorbed correlates with the level of stored iron. With depletion of iron stores, absorption of iron increases. Under special circumstances of excessive iron loss, intestinal mucosa may increase absorption of iron by 2–3 times more than normal (Finch and Cook 1984). Reinforcement of iron storage, in turn, leads to decrease in iron absorption.

The demand for iron varies significantly at different ages and between the sexes. The human fetus receives iron from the mother through the placenta. Under normal healthy development, an iron surplus accumulated in utero is sufficient for a doubling of the body mass after birth (Finch and Cook 1984). Thereafter, the child acquires iron from dietary sources and its amount and form become critical for rapid growth. Around 95% of iron necessary for production of hemoglobin in adult men comes from recycling iron and only 5% from the diet, while in a one-year-old infant around 70% of hemoglobin iron is reused from internal circulation and 30% comes from the diet (Dallman et al. 1980). In late childhood, requirement for iron is more balanced due to a more varied diet and a slowed rate of growth.

Starting from puberty male and female demands for iron differ significantly. Accelerating growth increases need for iron in both sexes, but females, due to

menstruation, require 50% more iron than males (Finch and Cook 1984). Physiologically, an adult male is in an advantageous position, as he requires only around 1 mg of dietary iron for homeostasis (Finch and Cook 1984). In the adult female, pregnancy is an additional factor that significantly increases demands for iron. During the last trimester of pregnancy the mother requires 5 to 6 times more iron than an adult man (Callender 1985). Apparently, females and children, especially young children, are the groups most at risk to develop iron deficiency anemia.

Influential Factors. The iron intake from the diet varies considerably depending on the form of the iron and combination of the foods consumed. There are two forms of food iron: the most common non-heme form of inorganic iron and heme iron present in meat (Rifkind et al. 1986). Because it is readily inhibited by other compounds in the diet, the absorption of non-heme iron is more complicated and restricted than that of heme iron. Among those dietary compounds that inhibit iron absorption are phosphates and phytates that are present in cereal and eggs; polyphenols from tea, vegetables, and legumes; and calcium in the form of cheese and milk that form insoluble solutions that reduce absorption of non-heme iron (MacPhail and Bothwell 1992). At the same time, ascorbic acid is a powerful enhancer of absorption of non-heme iron (MacPhail and Bothwell 1992). Although it is more rarely available, heme iron is absorbed easily and is assimilated intact by intestines, thereby avoiding negative influences of constituents that influence non-heme iron (Dallman et al. 1980). Meat also promotes absorption of non-heme iron from food (MacPhail and Bothwell 1992). Thus, the absorption of iron from

the diet strongly depends on the relationship between the form of iron available and other components in the diet.

Depletion of iron through blood loss is often considered the dominant factor in the development of iron deficiency anemia (Hoffbrand and Pettit 1993; Rifkind et al. 1986). Among the important routs of blood loss are menstruations in women and through the gastrointestinal tract in both sexes. On average, women lose approximately 40–80 ml of blood at each cycle (Hoffbrand and Pettit 1993). To maintain the balance, iron lost with blood should be replenished through the diet. Because in many cases the requirement for iron is not met by the diet, iron deficiency anemia is the most common type of anemia in women of reproductive age (Uthman 1998). Also, gastrointestinal blood loss due to infestation with parasitic worms is considered to be another major cause of iron loss in some developing countries (MacPhail and Bothwell 1992). Iron loss due to bleeding caused by gastric diseases, such as peptic ulcer, hiatus hernia, and carcinoma, although always considered in clinical practice, occurs with relatively low frequency (MacPhail and Bothwell 1992).

Anemia of Chronic Disease

Biomechanism. The anemia of chronic disease is anemia that occurs due to chronic illnesses, especially those of inflammatory nature (Uthman 1998). It is the second most common type of anemia after iron deficiency anemia (although some authors give more attention to the anemia of chronic disease considering that, because of minutia of iron required from dietary intake, iron deficiency can hardly develop due to poor diet solely; see Kent 1992). The mechanism of anemia of chronic disease is

different from that of iron deficiency and results from the body's natural self-defense from pathogens. Many pathogenic microorganisms invading the human body do not have their own iron store and, thus, rely on host iron for their replication. By binding free serum iron with storing protein and decreasing iron absorption from the food, the human body minimizes iron available for the pathogens (Weinberg 1992). This results in accumulation of storage iron and lack of iron for hemoglobin synthesis, which leads to anemia. Other characteristics of the biomechanism of anemia of chronic disease include decreased production of erythropoietin by kidney and erythroblast responsiveness to erythropoietin, which occur due to a growth-inhibited property of limphokines involved in inflammatory/immune response (Uthman 1998).

The relationship of anemia of chronic disease to development of porotic hyperostosis is a controversial topic. It is considered that short-term hypoferremia, resulting from an iron withholding mechanism, does not compromise iron metabolism (Stuart-Macadam 1992). Thus, the iron withholding mechanism is seen as a positive, adaptive response to pathogens (Stuart-Macadam 1992, 1998; Weinberg 1992). Other authors, however, considering the important role of iron in immune responses of the body to infectious diseases, argue that there may be a limit beyond which prolonged iron withholding increases rather than reduces susceptibility to pathogens (Cook 1990; Martin et al. 1985).

With the mechanism of iron withholding, anemia develops in a period of about six weeks (Rifkind et al. 1986). Clinical data show that even in mild form of anemia of chronic disease, 30% of patients demonstrate compromised iron metabolism with

production of hypochromic and microcystic red cells (Rifkind et al. 1986). Also, red blood cell life span is decreased to 60–90 days compared to a normal span of about 120 days (Rifkind et al. 1986). Under these circumstances, bone marrow usually increases production of new red cells, which, consequently, leads to expansion of bone marrow and development of porotic lesions. On the contrary, in anemia of chronic disease, the rate of production of red blood cells remains normal (Uthman 1998). Klepinger (1992), thus, stated that anemia of chronic disease would not produce porotic hyperostosis.

Influential Factors. The anemia of chronic disease develops from various conditions, including chronic infectious diseases, disorders of connective tissue (such as rheumatoid arthritis), and tumors (Rifkind et al. 1986). It is also can develop after extensive trauma or surgery and accompany other chronic anemias associated with liver or renal diseases (Rifkind et al. 1986). Anemia of chronic disease is considered to be mostly a mild and non-progressive kind of anemia, in which degree of severity relates to the severity of underlying disease (Hoffbrand and Pettit 1993). This anemia does not require specific treatment, should not be treated with supplemental iron, and can be corrected by treatment of underlying causative disease (Hoffbrand and Pettit 1993; Uthman 1998). Among characteristic features common for anemia (fatigue, shortness of breath, etc.), anemia of chronic disease is often accompanied by inhibited growth (Shin and Bellenir 1999; Uthman 1998).

Anemia of B12 and Folate Deficiency

Biomechanism. Vitamin B12 (cobalamin) and folate are two vitamins that play essential roles in production of blood cells. Their deficiency produces megaloblastic anemia, which is, by definition, the anemia that results from retarded synthesis of DNA in red cells at the stage of their formation in the marrow (Uthman 1998). While red cells in the circulating blood do not have nuclei, their precursors in the bone marrow, erythroblasts, have nuclei for reproduction by division. The role of folate is to transfer methyl groups to formation of thymidylate, one of the four nucleotides of DNA. B12, in turn, helps to clear methyl groups from the folate molecule to enable it to participate in further steps of DNA synthesis. Thus, with absence of either B12 or folate, DNA cannot be made (Uthman 1998).

Morphological alteration of the blood due to deficiency of B12 or folate is identical (Rifkind et al. 1986). A lack of either of them results in morphologic anomalous changes that are referred to as nuclear-cytoplastic dissociation, leading to a primitive immature cell nucleus and enlarged size of the erythroblast (Rifkind et al. 1986). In this condition many newly formed cells fail to divide and mature. Since maturation of the red blood cell is a requirement for it to leave the marrow and go into circulation, many cells die in the marrow. The outcome of this pathological process is anemia due to lack of new red cells in the blood and accumulation of erythroblasts in the marrow due to ineffective erythropoiesis (Uthman 1998). In addition, the production of other cells in the marrow such as platelets and granulocytes are affected as well. For all types of cells, decreased output into circulation occurs with increased production of their

precursors by the bone marrow, which in severe cases leads to hypercellular marrow (Rifkind et al. 1986).

Influential Factors. There are several ways by which anemia of folate and B12 deficiency can develop. Generally, in both types of deficiency, anemias developing from either malnutrition or malabsorption are recognized. However, specific ways and conditions of development of each anemia are different.

The most common cause of folate deficiency is malnutrition, especially in groups with increased demands: pregnant women and infants (Hoffbrand and Pettit 1993). Daily folate requirement, which is doubled in pregnancy, can be replenished through dietary components of food, of which the richest in folate is liver, but for most people usually comes from green vegetables. Anemia of folate deficiency can also occur due to malnutrition in cases of disorder of the upper part of the small intestines, where folate is absorbed. For instance, a gastrointestinal infection such as the protozoan parasite, *Giardia lamblia*, causes a condition known as "post infective malabsorption" (Callender 1985). Before the era of antibiotics in some tropical and subtropical regions throughout the world (India, Far and Middle East), a post-infective disorder of tropical sprue that is accompanied by diarrhea presented a serious threat, often with fatal outcome (Callender 1985).

Because stores of folate in the body are very limited and can be depleted in 3–4 months, the onset of folate deficiency is acute (Callender 1985). Reduction of the number of white cells and platelets leads to occurrence of ulcers in the mouth and on the

tongue as well as bleeding into skin from small capillaries (Callender 1985). Insufficient intake of folate during pregnancy can result in severe abnormality of the fetus, most especially neural tube defects (Uthman 1998). In infancy, the need for folate is 5–10 times greater than that of an adult (Uthman 1998). Pregnant women and infants constitute the main risk group for developing folate deficiency.

Unlike folate deficiency, deficiency of B12 rarely occurs solely due to dietary inadequacy nowadays. Although B12 is present only in food of animal origin, it is prevalent in many products, the daily requirement is minute, plus, the human body is good at holding accumulated cobalamin. The risk group for potential dietary deficiency consists mostly of strict vegetarians, especially babies breast-fed by vegetarian mothers; these infants may develop megaloblastic anemia with permanent neurological damage before it can be diagnosed (Uthman 1998). On the other hand, because the absorption of B12 is more complicated than that of other nutrients, its deficiency more commonly results from malabsorption. For successful absorption, the intrinsic factor should be secreted in the upper stomach and combine to form one complex with B12 and then move to the lower part of the small intestine where it will be absorbed. The most common causes of malabsortion of B12 and subsequent anemia include: chronic gastric disorders that lead to diminishing production of the intrinsic factor, or atrophic gastritis and regional enteritis in the jejunum or ileum, autoimmune disorder, and post infective syndrome of tropical sprue (Callender 1985; Rifkind et al. 1986). The most severe form of vitamin B12 deficiency due to lack of intrinsic factor is pernicious anemia, which is characterized by damage to the nervous system and was fatal before the investigation of

the cause at the beginning of the 20th Century (Uthman 1998). And finally, B12 deficiency occurs as a result of bacterial overgrowth or infestation by intestinal parasites such as fish tapeworm (*Diphyllobothrium latum*) that may grow 3–10 meters long (Rifkind et al. 1986). In both cases, consumption of B12 by these competitors leads to megaloblasic anemia of the host.

Accumulated stores of B12 in the body and a small daily requirement can prevent development of cobalamin anemia for up to 3–5 years, even without adequate replenishment from the diet (Uthman 1998). Possible symptoms at the onset of B12 deficiency are soreness of the tongue, which becomes red, smooth, and loses papillae, accompanied by slight jaundice. About 50% of patients demonstrate neurological involvement, which occurs due to the critical role of B12 in maintenance of neurons (Rifkind et al. 1986). With development of anemia, peripheral neuropathy appears first in long appendicular axes (arms, legs) with sensation of "pins-and-needles" and difficulties during manual work and walking; eventually the individual may become paralyzed. In some cases, mental illness develops with extreme expression of schizophrenic behavior, which is called megaloblastic madness. Nowadays, B12 anemia rarely occurs before age 40 and classically it is considered to be a disorder of elderly people (Uthman 1998). Since the cause is often the result of untreatable disorders related to intrinsic factor, the treatment of cobalamin deficiency continues for the rest of the individual's life. Even in the case of early diagnosis and treatment, the damage occurred to the nervous system is irreversible (Uthman 1998).

Studies on Archaeological Populations

Malnutrition. Numerous paleopathological studies refer to malnutrition as one of the main factors that caused iron deficiency anemia in past populations (El-Najjar et al. 1975; Palkovich 1987; Salvadei et al. 2001). A diet dependent on maize as a staple has been associated with increased prevalence of iron deficiency. El-Najjar (1975) examined 539 crania from prehistoric sites in the American Southwest. He found that 34% of all crania displayed porotic hyperostosis. The author pointed out that only around 5% of total iron contained in relatively poor-in-iron maize was absorbed. Maize also influenced iron intake from other kinds of food consumed simultaneously. Sites from two types of environment with different subsistence patterns were compared. It was observed that porotic hyperostosis was relatively uncommon in sage plains groups who had a variety of animal foods to complement maize. At the same time, heavily maize-dependent populations from the canyon bottom sites had much higher frequencies of porotic hyperostosis.

High frequency of porotic hyperostosis was also recorded in populations with subsistence based on various cereals such as wheat and rice (Lovell 1997; Pechenkina et al. 2002; Salvadei et al. 2001). Salvadei and colleagues (2001) suggested that poor nutrition with reliance on cereal as a staple may explain the high prevalence of porotic hyperostosis of 32.3% for the Lucus Feroniae population from 1st–3rd centuries AD and 41.5% for the Selvicciola population from 7th Century AD (central Italy). The authors speculated that iron deficiency in these populations might be triggered because of, first,

insufficient intake of heme iron and, second, the presence of phytates and fibers in cereals, leading to low absorption of iron from other foods taken with cereal.

Folate Deficiency. In recent studies of porotic hyperostosis found in ancient populations, anthropologists started to consider megaloblastic anemia as a causative factor in development of porotic lesions (Fairgrieve and Molto 2000; Sullivan 2005). For instance, Fairgrieve and Molto (2000) examined cases of cribra orbitalia in a skeletal sample of 385 individuals from Dakhleh Oasis, Egypt. They found that 67% of the examined populations from pre-Roman and Roman times were affected. Differentiation of the results by age cohorts revealed the highest percentage of active lesions in the age group of 0–6 months. Analysis of the infants' diet through the evidences from historical writing sources showed that the infants could be fed with goat's milk as a substitute for breast milk and weaned on goat's milk mixed with bread crumbs and honey. Goat's milk is known to be poor in cobalamin and folate compared to human milk. The authors pointed out that clinical research also shows that infants that are fed with goat's milk as a substitute to mother's milk develop a severe megaloblastic anemia at age 3–5 months. Thus, it was suggested that the folate deficiency and subsequent megaloblastic anemia was the main cause of the porotic lesions of the orbits in the pre-Roman and Roman populations of Dakhleh Oasis in Egypt.

Intestinal Parasites. Bioarchaeological studies present evidences that link helminthes to the occurrence of porotic hyperostosis as a skeletal expression of anemia in

past populations. For instance, Sullivan (2005) analyzed prevalence and etiology of anemia in individuals of different social status from Medieval York. The author suggested that parasitic infestation was one of the key factors in developing anemia among all social groups. She argued that, while anemia due to poor diet or chronic infectious diseases might be other causes of anemia in the low status group, intestinal parasites are the only suspected cause of anemia for upper social classes with a prevalence of 29.4% in religious males and 33.3% in high status males and females. Several types of intestinal parasites, such as whipworm (*Trichuris trichiura*) and roundworm (*Ascaris lumbricoides*) that were found in archaeological remains from Medieval York support the author's suggestion that parasitic infestation was a major problem in the Medieval period.

One of the most detailed and convincing studies of the role of intestinal parasites on the etiology of porotic hyperostosis was presented by Reinhard (1992). The author analyzed coprolites from several horticultural sites of the American Southwest regarding helminthes and arthropod infections, and floral and faunal components. Reliance on maize as a staple and low consumption of meat was revealed by analyses. The sample also exhibited diversity of helminthes presented by hymenolepidids, *Strongyloides, M. clarki, tremonode, A. lumbricoides, T. trichiura*, and especially high index of *E. vermicularis* (pinworms). The tests for correlation of prevalence of porotic hyperostosis with maize and meat consumption brought negative results. At the same time, correlations of all helminthes with porotic hyperostosis and pinworms with porotic hyperostosis were significant at the 95% confidence limit. It was pointed out that

pinworm is linked to microparasitic infections that caused diarrhea and other infectious diseases, which may be considered the direct causes of anemia.

Infectious Diseases. Numerous studies demonstrate a positive correlation between porotic hyperostosis and periostitis (bone marker of infectious diseases) indicating the relationship of infectious diseases and anemia (Grauer 1993; Lallo et al. 1977; Larsen and Serring 2000). In this regard Sullivan (2005) speculates that, due to the catalytic role of iron in immune responses, the mechanism of iron withholding that leads to anemia of chronic disease serves as a defense against infectious diseases but only to a certain point beyond which severe and prolonged hypoferremia predisposes an individual to infectious diseases. In her study of acquired anemia in Medieval York (England), Sullivan (2005) inferred that due to chronic exposure to pathogens, anemia of chronic disease was one of the significant factors that influenced high prevalence of cribra orbitalia among individuals of low social status.

Blom and colleagues (2005) suggested that individuals may become anemic due to infectious diseases because 1) gastrointestinal infections affect the host in the same way as macroparasites, and 2) bacteria deplete the host's iron by using it for their own replication, and 3) the relationship of anemia to chronic diseases. In the study that included 1,465 individuals excavated at various sites in Peru, these authors tested the hypothesis of prevalence of porotic hyperostosis due to such environmental stressors as diet, parasitism, and infectious diseases. Comparison of the distribution of cribra orbitalia between populations that subsisted on marine diet and populations with a maize-

based diet did not support the hypothesis of association of anemia with dietary stress. As it was predicted based on archaeoparasitological data, lower frequency of the lesions was found in the sites at lower altitude, closer to the coast, and in the northern, less arid part of Peru. At the same time, heightened mortality associated with a lower frequency of porotic lesions indicated the importance of other environmental stresses. The prevalence of cribra orbitalia in less arid environments led the authors to the conclusion that water-borne tuberculosis or tuberculosis-like pathogens were the major causes of anemia in these populations (Blom et al. 2005).

Sex Differences. Examination of sex differences in the prevalence of porotic hyperostosis and cribra orbitalia has its limitations in archaeological context related to problems with sexing subadults. Consequently, the results of studies of sex differences are biased toward analysis of adults. In agreement with contemporary clinical data, many anthropological studies observed that females were more often affected than males (Cybulski 1977; Salvadei et al. 2001; Slaus 2000; Stuart-Macadam 1998). For instance, Cybulski (1977) examined frequency of cribra orbitalia in the sample from the British Columbia coast and found that females were affected almost three times more frequently than males (13.3% in females versus 4.8% in males). Females' susceptibility to iron deficiency anemia was attributed to iron loss during menstruation, multiple pregnancies, and breastfeeding.

Other studies found higher frequency of porotic lesions in males (Danforth et al. 1997; Mittler and Van Gerven 1994). Danforth and colleagues, for example, investigated

porotic lesions among colonial period Maya from Tipu, Belize. They found that males had significantly higher prevalence of porotic hyperostosis 24% compared to females who had only 11.6%. Males were also more frequently, although not significantly, affected by cribra orbitalia, 8.8% versus 5.4% in females. At the same time, ethnographic records present evidences of preferential treatment of males in Maya culture. The authors concluded that despite the possible preferential treatment, males were not able to overcome the advantages of genetic buffering that females naturally have and, thus, males experienced less healthier childhood. Many other anthropological studies did not observe sex differences in the prevalence of porotic hyperostosis and cribra orbitalia (El-Najjar et al. 1976; Walker 1986).

Correlation with Longevity. Anthropological studies that examined the impact of anemia on overall health and longevity mostly focused on analysis of survival and life expectancy of affected individuals versus non-affected. Mittler and Van Gerven (1994) performed demographic analysis of cribra orbitalia among a Medieval population from Kulubnarti (Nubia). Life table data clearly demonstrated that at age 5, individuals with porotic lesions had a mean life expectancy 15.5 years lower than individuals without porotic lesions. The prevalence of cribra orbitalia in those who died before age of 13 is 70%, while among older age groups it is 30% on average. The authors concluded that there might be various factors that contributed to an individual's death, but anemia was one the major factors influencing life expectancy in the Kulubnarti population from the early Christian period.

Also, Blom and co-workers (2005) examined the impact of anemia on the health and mortality pattern among the coastal populations from pre-Columbian Peru. Age analysis showed that porotic lesions were significantly prevalent among individuals who died in childhood (81.8%), while prevalence among those who survived to adulthood is 19% in females and 18.9% in males. To demonstrate the impact of anemia on overall health and life expectancy, the authors calculated the ratio of lesion frequency between those who died in childhood and those who survived to adulthood. The sample from the Central coast showed the ratio of 3.5:1, while the South Central and South samples had ratios 2.1:1 and 2.2:1 respectively. Thus, Blom and colleagues concluded that anemic children from the Central coast population had lower chances to survive to adulthood than their peers from other investigated regions.

Summary

Porotic lesions of the skull vault and eye orbits observed in skeletal remains have been associated by most anthropologists with anemia. Anemias due to iron, vitamin B12, and folate deficiencies are named among the most probable causes as deficiency in any of these nutrients leads to increased erythropoesis, which, in turn, eventually results in porotic lesions. According to clinical descriptions, anemia of chronic disease is not characterized by increased erythropoesis. It is, however, speculated in anthropological literature that chronic exposure to pathogens and prolonged iron starvation due to the iron withholding mechanism, would eventually trigger increased erythropoesis and lead to porotic lesions. Thus, infectious diseases, inadequate nutrition that lacks iron, vitamin B12, or folate, and blood loss due to intestinal parasites are considered to be among the

main causative factors leading to development of anemic conditions. Because of the similarity of bony expressions of different kinds of anemia and non-anemic causes of porotic lesions such as scurvy and inflammatory reaction, it is important to consider different factors that might be involved in the development of porotic hyperostosis.

Periostitis

Osteitis is an inflammatory reaction of bone to infections. There are two descriptive terms, osteomyelitis and periostitis, that refer to inflammatory responses of bone to infections. Osteomyelitis is the pathological process characterized by involvement of bone marrow, production of pus, bone destruction, necrosis with formation of the sequestrum, and simultaneous apposition of new bone, forming an involucrum (Roberts and Manchester 1995). The inflammatory reaction of the fibrous layer of periosteum, called periostitis, is characterized by the presence of a new woven bone formation over the cortical bone that subsequently may be reorganized into lamellar bone and is seen as irregular thickening of the bone surface (Ortner and Putschar 1985)

Biomechanism of Inflammatory Reaction of Bone Tissue

Infection can affect bone either via hematogenous spread from primary focus or by direct trauma. In case of hematogenous proliferation of pathogens, the most susceptible part of the long bone in infants and children is usually the metaphysis because of the intensive blood supply of this area during the growth process. In infants, infection invades through the growth plate of the metaphysis and often envelopes the adjacent

joint, while in older children and adults, the metaphysis is as often involved as the diaphysis (Aufderheide and Rodriguez-Martin 1998).

Over the lifetime, bone is covered with a thin layer of fibrous connective tissue that is called periosteum. Underneath the periosteum are situated osteoblast cells responsible for bone formation and maintenance (White 2000). Pathogens penetrate the thin membrane of periosteum and reach cortical bone. Inflammatory reaction and subsequent abscess formation lift the periosteum from the bone cortex (Steinbock 1976). Separation of the periosteum triggers osteoblasts to form new bone in attempt to enclose the affected area (Aufderheide and Rodriguez-Martin 1998). New woven bone that lies over the involved portion of the bone cortex (involucrum) represents periostitis.

In case of osteomyelitis, spreading infection proliferates through the bone cortex into the medullary cavity. To reach the cavity, pathogens follow Haversian and Volkmann canals that are hollow passages for blood, lymph, and nerve fibers of the cortical bone. Infection of blood vessels that lead to vascular obstruction and extensive new bone formation induce ischemia (reduced blood flow). Subsequent necrosis of some portions of the cortical bone creates sequestra (areas of dead bone). Eventually, abscess perforates the involucrum forming cloacae (drainage channel). Sequestra and cloacae are characteristic features of osteomyelitis (Roberts and Manchester 1997). In traumatic cases, infection can directly reach the medullary cavity.

Etiology

Infectious diseases generally can be divided into four groups: bacterial, viral, fungal, and parasitic. The majority of cases of periostitis found in skeletal remains are

attributed to chronic bacterial infections (Goodman and Martin 2002). Acute infections caused by viruses usually are short-term, fast resolving or fatal and, thus, do not leave marks on bones (Goodman and Martin 2002). Fungal infections affect bones secondary to haematogenous dissemination, but they commonly constitute a small percentage of bone infections (Aufderheide and Rodriguez-Martin 1998). Some parasitic infections, mostly of helminth nature, which contribute to iron or vitamin deficiency, may indirectly cause bone lesions of specific pattern of porotic hyperostosis (Stuart-Macadam 1992).

Contemporary clinical data suggest that *Staphylococcuc auerus* is the most commonly observed bacterium in the world involved in nearly 90% of cases of osteomyelitis, while *Streptococcuc pyogenes, Haemophilus influenzae, Bacillus coli,* and other bacteria such as pneumococcuc, meningococcuc, and so on are recorded in about 10% (Aufderheide and Rodriguez-Martin 1998). There are two main ways for bacteria to enter the body. The most common is the haematogenous spread from the primary sites of infection, which usually are tonsils, middle ear, and bronchial tree (Roberts and Manchester 1997). The more occasional second way is the direct infection from the skin or bone injury. In most of the cases, the infections brought by various micro-organisms produce identical non-specific pathological changes reflecting a basic inflammatory component of immune response to an invading pathogen.

There are several types of infectious diseases, such as those caused by *Mycobacterium tuberculosis* and *leprae*, and *Treponema pallidum* manifested in yaws and syphilis, that produce distinctive diagnostic symptoms in affected individuals (Ortner and Putschar 1985). These specific types of infection leave characteristic patterns of

bone lesions that are not simply determined but can nevertheless often be recognized through careful differential diagnosis (Hershkovitz et al. 1998). Pulmonary tuberculosis is transmitted through exhaled droplets, while leprosy is transmitted both by skin contact and through inhalation from an individual with severe nasal infection; yaws and endemic syphilis is transmitted by bodily contact, and venereal syphilis is transmitted through sexual intercourse (Roberts and Manchester 1997).

Influential Factors

Resistance to various infections depends on the virulence of the infectious agent, host characteristics and environmental influences (Scrimshaw et al. 1968). One of the laboratory experiments with mice vividly demonstrated interaction between these factors (Scrimshaw et al. 1968:145). In that study, three genotypes of mice (specifically inbred resistant to diseases, inbred susceptible, and non-selected outbred group) were exposed to virulent, avirulent, and mixed type of pathogens and fed an adequate and an inadequate diet. The results revealed that when the virulence of the infectious agent was high, all groups succumbed to disease despite the type of genotype or diet. When the pathogen was avirulent, all groups survived regardless of genotype and type of diet. All three groups, however, demonstrated different results with the mixed type of pathogens. In the group of resistant genotype, all hosts survived regardless of the type of diet, while in the susceptible group, all hosts died regardless of the diet, which clearly demonstrates an important role of the host characteristics in resistance to diseases. The only group that showed a significant role of diet was the group of non-selected outbred mice exposed to mixed type of pathogens. In this group, only those who received an adequate diet

survived. Importantly, according to Scrimshaw and colleagues (1968), this last experimental combination represents a real-life situation with normal distribution of individual resistance to diseases and various degree of the virulence of pathogens. Thus, host characteristics (such as nutritional status, genetic factors, etc.) are significant factors that determine outcome of infectious diseases.

Role of Immune Responses. The function of immune response is a complex mechanism that involves interaction of multiple organs and subsystems. Functional immune response emerges during the fetal stage and gains immunocompetence in later development (Kimball 1986). In general, the immune responses to infections engage three components of defense system: non-specific defense, humoral-mediated defense, and cell-mediated defense.

The non-specific component represents a first line of resistance that provides protection against pathogens without recognizing them (McDade 2003). The non-specific component includes various factors, among which phagocytic cells are the most powerful and critical force of the defense system. Both types of phagocytic cells, polymorphonuclear leukocytes (70% of which are neutrophils) and macrophages first appear in bone marrow (Mims 1976). The inflammatory response to bacterial infection begins with an acute phase that is characterized by vascular reaction (Ortner and Putschar 1985). Leukocytes are released by capillaries from the circulatory system and migrate to the bacterial focus. Production of pus marks the site of phagocytosis of bacteria by leukocytes.

Humoral-mediated defense mechanisms recognize and eliminate antigens on extra-cellular level (McDade 2003). B-lymphocytes and antibodies are characteristic components of humoral defense. Antibodies, which represent five main immunoglobulin classes (IgA, IgD, IgE, IgG, and IgM), are produced by B-cells in cooperation with T-cells as immune responses to specific antigens (Kimball 1986). The process takes place mostly in the lymphoid system and also in respiratory and gastrointestinal tracts. The production of antibodies in response to most of the infections encountered during the lifetime reflects the natural process of immunization (Mims 1976).

Specific immunity, including T- and B-lymphocytes, is primarily a defense that can recognize and target specific antigens. Some of the pathogens are intracellular organisms, which survive phagocytosis, continue to replicate within macrophages, and cannot be reached by antibodies. Only T-lymphocytes have the ability to specify and target antigens on intra-cellular level (McDade 2003). These lymphocytes are involved in defense against most viruses, fungi, and some bacteria (such as mycobacteria) (Edelman 1977).

Immune responses represent interaction of both cell-mediated and humoral defences. Lymphocytes and plasma cells are involved in the process during the sub acute and chronic phases of inflammatory reaction to bacterial infection (Ortner and Putschar 1985). Also, both humoral and cellular components of immune responses are present in immune response to viral infections. Lymphocytes and macrophages are present in cellular response while neutrophiles are not. Consequently, viral infections are not usually represented by pus (Ortner and Putschar 1985).

Role of Nutrition in Host Resistance. The essential role of diet is recognized because of the importance of various proteins, amino acids, and vitamins and minerals for the proper functioning of immune responses. One of the reasons for this is the high energetic requirement of immune defenses (McDade 2003). An increase in body temperature by one degree of Celsius requires a 13% increase in metabolic rate (McDade 2003:104). Malnutrition has been shown to have negative effects on all aspects of immune response. Clinical studies found that cellular defenses are especially sensitive to insufficient nutrition. Moderate and severe malnutrition leads to atrophy of the thymus, reduced number of T-lymphocytes, and, consequently, delayed responses to viral and bacterial infections, such as *Herpes simplex* and *Mycobacterium tuberculosis* (Petro et al. 1984).

Primarily B-cell/antibody defenses are better protected from mild and even severe malnutrition. Experimental studies showed that responses to specific bacterial pathogens expressed in B-lymphocytes and immunoglobulin levels often remain normal or elevated in malnourished animals (Petro and Bhattacharjee 1981). The results, however, are inconsistent. Chandra (1975), for example, found significant reduction of IgA in response to polio and measles vaccines in moderate and severely malnourished individuals.

Essential nutrient deficiencies can affect non-specific defenses. Some proteins, specifically of plant origin, lack amino acids that are necessary for adequate nutrition. For instance, maize proteins are scarce in lysine, while soy protein is limited in

methionine, and rice, in methionine and threonine (Petro et al. 1984). Other clinical and experimental studies were able to demonstrate that function of immune defenses could be affected by deficiencies in other necessary nutrients and microelements, such as vitamins A, B, and E, selenium, zinc, and iron (Bendich and Cohen 1988; Dallman 1987; Fischer Walker and Black 2004; Van Vleet and Watson 1984; Vyas and Chandra 1984). For example, Fischer-Walker and Black (2004) emphasized the importance of zinc in resistance to infectious diseases through the results of supplemental studies. It was shown that zinc supplementation positively influenced reduction of diarrhea (by 18%) and pneumonia (41%). They demonstrated that maternal supplementation with zinc may lead to a reduction in infant infections and a decrease in child mortality rate by about 50%.

Role of Genetics in Host Resistance. Host genetics is one of the factors that determine host resistance to infectious diseases. Numerous studies among monozygotic twins and families demonstrated a genetic contribution to susceptibility to infectious diseases (Abel et al. 1991; Malaty et al. 1994; Sorensen et al. 1988). For instance, Abel and colleagues (1991) analyzed response to *Schistosoma mansomi* among Brazilian families. They determined that a single codominant gene was responsible for the level of infection. It was later found that this gene is involved in encoding proteins that control T-cell immune function. As a result of genetic differences, 5% of the population was highly susceptible to schistosomiasis, 60% demonstrated resistance, and 35% showed intermediate reaction (Kimman 2001:64).

The interrelationship between infections and genetics is reflected in the important role of infections in natural selection and human evolution. Diseases that kill or contribute to the lower reproductive success of a population are likely selective agents (Van Blerkom 2003). "Infectious disease-driven selection may not only maintain diversity within populations, but also promote divergence between populations" (Van Blerkom 2003:35). Human blood groups and their spatial distribution over the world may serve as another example of evolutionary influences of infections on diverse genetic predispositions to various diseases. For instance, high frequency of blood group B in central and southern Asia is hypothesized to be the result of selective forces against group A, which shows low resistance to smallpox, and against group O, which demonstrates high risk of acquiring plague and cholera (Kimman 2001).

Genetics plays a significant role in molding resistance to certain infectious diseases. For instance, S, C, and E hemoglobinopathies, which affect up to 80% of some populations in the Mediterranean and many regions of Africa and Asia, are the result of evolutionary mutation to resist severe infection of malaria spread by *Anopheles* mosquitoes (Kimman 2001). These mutations of a single gene may lead to diseases incompatible with life, but more commonly are present in milder forms. As an advantage, carriers of hemoglobin S are found to have 90% resistance to severe malaria (Hill et al. 1991).

Role of Environmental Factors. Various environmental factors are crucial determinants of the distribution and prevalence of infectious diseases. Multiple

environmental factors are subdivided according to physical factors, which include geographic and climatic characteristics of the environment, biological factors, which include floral and faunal features, and socio-economic factors, which include human to human relationships (Fox et al. 1970). Evaluation of the environmental influences presents a challenge because of the involvement of various factors that simultaneously and often indirectly affect the infectious agent and host and the balance of relationship between them. Still, epidemiological analysis provides valuable information on key factors of occurrence and spread of infectious diseases.

Epidemiological studies show that many infectious diseases have a clear geographical association. Geographic distribution of diseases relates to the climatic and environmental habitat of infectious agents. Thus, spread of malaria in tropical regions of the world is restricted to favorable habitats for mosquitoes that serve as a vector in spread of malarial infection (Sallares et al. 2004). Another example is treponemal diseases. According to the Unitarian theory, the treponemal infections, such as pinta, yaws, non-venereal syphilis, and venereal syphilis, are not different diseases caused by distinct pathogens but a single disease (treponematosis) caused by slightly distinct strains of one agent, *Treponema pallidum* (Steinbock 1976). Climatic adaptation of the pathogen to various environmental conditions leads to different syndromes of treponemal diseases: whole body surface involvement in hot humid tropical climate (yaws); mouth, armpits, and crotch involvement in hot dry climate (non-venereal syphilis); and sexual organs involvement is more common in temperate regions (Steinbock 1976).

The biological characteristics of the environment, which include all living organisms—plants and animals, are a significant factor that indirectly or directly influences human health (Fox et al. 1970). Plants and animals that constitute the basis of human diet indirectly influence human health, determining nutritional status and, consequently, susceptibility to infectious diseases. Many infectious agents are zoonotic, that is they have the ability to cause disease in humans as well as in specific animal species, which serve as a basic reservoir for that agent. H5N1 avian influenza originated in domestic ducks and *Streptococcus iniae*, which can affect fish and humans, are vivid examples of zoonoses (Miller and Neely 2005; Sims et al. 2005). Some infectious agents are transmitted to humans by arthropod vectors (insects, arachnida, etc.). Malaria transmitted by mosquitoes and epidemic typhus transmitted by lice are infamous examples of infections transmitted by arthropods (Fox et al. 1970).

Proximity to animal hosts or vectors of infectious disease and consumption of meat or milk of affected animals contribute to the spread of infections from animals to humans. For example, Salzano (1990) analyzed the prevalence of *Taxoplasma gondii* among South American Indians. This parasite affects both humans and animals, but the mode of transmission is not clear. The author analyzed the results found in several studies and demonstrated that differences in prevalence between tribes can be attributed to dietary preferences. The economy of the tribe that showed the lowest frequency of infection focused on fishing, while the economies of tribes with a high prevalence of infection are based on hunting and animal husbandry. Thus, consumption of infected meat was suggested as one of the main factors in the spread of infection.

Socio-economical characteristics of the environment include population size and density; sanitation; level of economic, social, and scientific development; medical care; communication; and many other factors. Although these factors affect human health indirectly, they can play a crucial role in spread of infectious diseases. Jorde and co-workers (1990) compared the temporal and spatial pattern of death rate from smallpox between the mainland settlement of Kitee and settlements from the Aland Islands (Finland) based on the parish records from the two regions. The records demonstrated that both regions experienced the peak of death from smallpox during the springtime. In part, this was attributed to more frequent travel in spring, which provoked the spread of the virus. The researchers also found that the mainland settlement had significantly higher periodicity of smallpox epidemics (4.4 years) than island settlements (7 years). Analysis of two populations allowed the authors to relate higher periodicity of smallpox epidemics in the mainland parish to higher communication level through religious and marital practice. Population density, which was higher in the mainland parish (1.6–9.6 persons per square kilometer versus 0.6–2.0 p/km^2 in the island parishes), was another factor that influenced higher periodicity of epidemics in the mainland parish.

Studies on Archaeological Populations

Endemic Pathogens. Study of causative factors includes examination of possible pathogens and diseases responsible for periosteal lesions and analysis of various environmental and cultural factors that might influence morbidity of investigated populations. For instance, Buckley (2000) studied the pathological lesions observed on the cranium and postcranial remains of 17 subadults from pre-European burial mounds in

Tonga, Polynesia. Pathologies were recorded in 42–50% of the individuals who exhibited tissue resorption and subperiosteal new bone formation. The lesions affected mostly extremities of the tubular bones, entire diaphysis in some cases, and endocranial surface. After considering the pattern of lesions, their distribution within the skeleton, and the severity of involvement of different age groups, the author suggested that infectious diseases responsible for discovered lesions were yaws and weanling diarrhea aggravated by metabolic disturbances from scurvy and hypervitaminosis A. Congenital syphilis, Caffey's disease, and haematogenous osteomyelitis were ruled out through the differential diagnosis.

Malnutrition. While examining etiology of infectious diseases in archaeological populations, many authors emphasize the importance of the various background environmental and cultural factors of the investigated populations. Association between markers of infectious diseases and anemic lesions of porotic hyperostosis found in many studies allowed researchers to relate the prevalence of infectious diseases to malnutrition. Grauer (1993) studied patterns of anemia and infections in a sample of 1,014 individuals from Medieval York, England. Age distribution of the lesions revealed earlier onset of porotic hyperostosis relative to the occurrence of lesions of infectious diseases. The author suggested that poor nutrition and depletion of subadults' iron stores contributed to increased susceptibility to infectious diseases.

Lallo and colleagues (1977) also analyzed the association of the lesions of infectious diseases with anemic lesions in the sample of 269 individuals from Dickson

Mounds and Eiden in Lorain, Ohio. The results revealed occurrence of periosteal lesions in 73.8% of those individuals who had porotic lesions, demonstrating strong synergetic correlation between infectious diseases and malnutrition. Occurrence of periosteal lesions increased with transition from hunter-gatherer subsistence pattern (50%) to agricultural subsistence focused on maize (75%).

Population Size and Density. Lambert (1993) examined temporal changes in prevalence of periosteal lesions in the prehistoric populations from the Santa Barbara Channel Islands. The results showed a significant increase in frequency of periosteal lesions from the Early (6000–1400 BC) to the Middle (1400 BC– AD 1150) periods. The author emphasized that the subsistence pattern of the investigated hunter-gatherer populations was based on marine fishing and so was rich in animal protein. Thus, she suggested that increase in frequency and severity of lesions over time was related not to malnutrition but to living conditions favorable for spread of infectious diseases, such as sedentism and increased population size and density. Recognition of treponemal disease in two skeletal remains allowed the author to speculate that introduction of treponemal infection that was not present in the region previously also might account for increased prevalence of periosteal markers in the Middle period.

Correlation with Longevity. Some studies investigated the issue of association between markers of infectious diseases and longevity (Goodman et al. 1984b; Grauer 1993). The results, however, are controversial. Goodman and co-workers (1984) studied

the pattern of the markers of physiological stress in prehistoric population in Dickson Mounds, Illinois. They found that individuals without infectious markers demonstrated longer life span than individuals with infectious lesions. The mean age at death for individuals without tibial periostitis was 39.5 years, for individuals with slight periosteal lesions it was 37.1 years and for those with severe lesions it was 35.1 years. In contrast, Grauer (1993) noted an increase in mean age at death for individuals with markers of infectious diseases. Grauer argued that adults who survived childhood stresses had good immune responses to infectious diseases.

Sex Differences. Many authors examined sex differences in prevalence of infectious diseases (Bourbou 2003; Cassidy 1984; Larsen 1984; Slaus 2000). The results of these studies are inconsistent. Bourbou (2003), for instance, found that males were affected with periosteal lesions more frequently than females accounting for 57.1% in Messene and 71.4% in Eleutherna settlements from the Proto-Byzantine period in Greece. The author attributed the prevalence of periosteal involvement in males versus females to differential activities. According to the author's opinion, everyday activities that were performed mainly by males often resulted in trauma of soft tissue and consequent chronic inflammation of underlying bones. Conversely, Slaus (2000) found that females demonstrated more periosteal lesions. Slaus (2000) observed periostitis in 24.2% of females and 11.4% of males in the sample from Nova Raca (Croatia) dated to the late medieval period (14^{th}–17^{th} centuries). Also, females exhibited a higher percentage of active lesions at the time of death. The author speculated that the poor

health status of females from an early subadult age reflected a limited access to food related to a more disadvantageous social position as compared to males.

Summary

Infectious disease can be of viral, bacterial, fungal, or parasitic in nature. The majority of infectious diseases, especially those of viral origin, are restricted to soft tissues because they resolve quickly, either successfully or fatally, before infections can spread to the bone tissue. Chronic bacterial infections are the main causative factor of skeletal involvement. Periostitis and, in more severe cases, osteomyelitis are skeletal markers of chronic infectious diseases. Resistance to infections depends on the virulence of the pathogens and host characteristics. Infants and young children constitute the main group of risk for illnesses and death from infectious diseases due to immunoincompetence. Among the key factors of host resistance is adequate nutrition because the function of immune response, specifically T-cell defenses, is a costly physiological process that requires high energetic expenses and the availability of various proteins, amino acids, vitamins, and minerals. Also, epidemiological and anthropological studies demonstrate that environmental factors play an essential role in the distribution of infectious disease.

Stature

Stature (standing height) is a valuable parameter in assessment of human health. Studies of first and second degree relatives and twins demonstrate a significant genetic

effect on stature (Tambs et al. 1992). At the same time, numerous studies of living and archaeological populations show that height is sensitive to such environmental factors as diet and diseases (Eveleth and Tanner 1990; Maat 2005; Steckel 1995). The research emphasizes a positive correlation between living conditions during childhood and resultant adult stature (Crook 1994; Foster et al. 2005; King and Ulijaszek 1999). Ultimately, human stature is an outcome of an interrelationship between genetic and environmental factors that influence an individual from the earliest stages of growth and development (Eveleth and Tanner 1990). In attempts to gain information on health from stature, researchers have studied it from several perspectives, such as childhood growth profile, temporal fluctuation in adult stature, sexual dimorphism in stature, and correlation of stature and longevity.

Biomechanism of Growth

Recent genetic research suggests that hereditary influences on stature make up to 75–90% of observed variances (Silventoinen et al. 2000). Studies of genetically related people demonstrate the major contributory role of genes on stature (Bogin 1988; Tanner 1978). Due to differences in genotype, the pattern and tempo of growth (common for all humans in general outline) varies between individuals, populations, and sexes (Leigh 1996; Silventoinen et al. 2000). Genes also influence velocity and duration of growth as an individual attempts to reach his/her genetically predetermined height (Cameron and Demerath 2002; Waddington 1957). Thus, the influence of heredity on growth is evident in the variations of adult stature, pattern, velocity, and duration of growth between individuals and sexes.

Genetic Control of Height. The studies of families and, especially, twins present the most vivid evidence of the degree of genetic control over human stature. Genetically related individuals tend to resemble each other in height. For instance, Byard and colleagues (1983) analyzed data from a longitudinal study of familial correlations in height. The data included measurements that were taken from relatives with varied degrees of genetic relationship (siblings, parents and offspring, uncles and nephews, etc.) once a year from 1 to 18 years of age. The results revealed that variation in stature correlated with degree of relatedness (Byard et al. 1983). Eveleth and Tanner (1990) also showed that, while height of males from one population varied up to about 25 cm, in a family between brothers and sisters the range was about 16 cm, and for monozygotic twins, who share the same genotype, it was just 1.6 cm. In other words, because of significant genetic control of height, the closer the genetic relatedness, the more similar adult stature.

Pattern of Growth. Human growth follows a pattern that is unique among mammals (Bogin 1988). Due to genetic regulation, all humans conform to a pattern of intensive growth during the fetal stage and early childhood followed by a period of slow growth in middle childhood, and then another growth spurt in adolescence (Leigh 1996). Bogin (1988) compared the growth chart of French children from the 18th Century to a chart documenting height measurements of U.S. children. He found the charts closely resembled each other and differences in height increase were not statistically significant

in any age period. The author summarized that, in general outline, the pattern of growth is similar for all normal children.

Growth Timing. There is variation among individuals and populations in the timing of initiation and duration of growth spurts due to genetic differences. Tanner (1978), for instance, demonstrated that monozygotic twins who grew up in the same environment had growth schedules differing by only two months, while dizygotic twins had a growth pattern differing, on average, by twelve months. The timing of growth also differs among populations. Eveleth and Tanner (1990) compared the growth curves of descendants from three geographically separated populations including those with ancestors from Africa, Asia, and Europe. They found that groups with African ancestry had the earliest onset of an adolescent growth spurt, followed by Asian descendants, and, lastly, those of European descent. At the same time, Asians showed the lowest velocity of growth compared to two other groups (Eveleth and Tanner 1990).

Moreover, there is a genetically predetermined difference in growth timing between sexes. At a point half way through fetal development, females are 3 weeks more mature than males, while at birth the difference is 4–6 weeks, and at puberty, it increases to 2 years (Tanner 1978). The general trajectory of growth is similar, but girls enter growth spurts earlier than boys. Feldesman (1992) analyzed subadult stature in different age groups from 8 to18 years of age. He traced identical trajectories of growth for both sexes. Girls, however, exhibited their adolescent growth spurt around age 10–11, with a peak at 12, while boys entered the spurt around 12 years of age and had a peak around 14.

Canalization. A characteristic feature of the growth process is the phenomenon of canalization—the tendency of growth to return to its original path (channel) if circumstances have pushed it off course (Waddington 1957). Tanner (1978) noted, for example, that lengths of bodies of monozygotic twins are different at birth. He showed that the monozygotic twin, who had suffered unfavorable conditions *in utero*, had reverted to a normal trajectory of growth after birth. Under favorable conditions, catch-up growth (higher than normal velocity of growth) can restore the height to its genetically predetermined level (Sandige et al. 2004). Various studies on living populations show, however, that if favorable periods are short, full catch-up growth may never be complete and stunting would be permanent (Crook 1994; Foster et al. 2005). Delayed maturation and a prolonged period of growth are the other ways to reach the genetically programmed target (Tanner 1978). Apparently, there are two secular trends of growth traced at a population level: "positive" with a reduced growth period that results in increase of average stature (advanced maturation) and "negative" with a long growth period that often results in decreased average stature (delayed maturation) (Maat 2005).

Influential Factors

Environmental factors considerably affect growth and resultant stature. Nutrition, infectious diseases, and their interplay are the key environmental factors that interfere with human stature (Eveleth and Tanner 1990). Adverse living conditions that compromise health during childhood may impede growth rate and lead to permanent stunting (Foster et al. 2005). Thus, the differences between individuals within

populations and between populations can be explained in part by different environmental influences (Cook 1984; Genoves 1967; Walker and Thornton 2002).

Role of Nutrition. Diet is one of the most important components for healthy growth and development. The multiplication and growth of cells requires adequate amount of protein, lipids, vitamins, and minerals, approximately 48 of which are essential for human growth and have to be acquired from food (Bogin 1988). Malnutrition negatively affects normal growth. For this reason, researchers consider growth as an indicator of nutritional status (Bogin 1988; Shanklin 2000). By definition, malnutrition may imply starvation (that is, almost complete absence of food), under-nutrition (an inadequate quantity of food), specific deficiency (lack or limitation of specific nutrients), and imbalance (i.e., a disproportion of nutrients) (Scrimshaw et al. 1968).

The total amount of calories consumed is one of the major components of diet that influences human growth. Studies of living populations suffering chronic under-nutrition demonstrated a delay of childhood growth and shortened stature (Crooks 1994; Hodge and Dufour 1991). For instance, Garn and Clark (1975) analyzed data from a large survey of nutritional adequacy performed in the United States in the 1960s and revealed that children from lower income groups were shorter than children from higher income families. The authors emphasized that all groups had necessary nutrients in their diet, but those from low-income families received less total calories, which resulted in a growth deficit.

Protein is one of the most essential dietary compounds needed for normal growth. Dietary protein supplies amino acids, required for the formation of new tissues and the increase in nitrogen concentration (Gopalan and Rao 1979). Experiments with rats have demonstrated that protein deficiency, even when the total caloric intake was adequate, prevents synthesis of the insulin-like growth factor, which stimulates somatic cell growth and division as well as bone, and cartilage growth (Prewitt et al. 1982). Reports from developing countries indicate that death from kwashiorkor, one of the severe forms of protein deficiency that is characterized by poor growth, anemia, weakness, and edema, may occur in a few weeks after appearance of clinical signs (Gurney 1979).

Vitamins D, C, and A are among those that have been found to influence growth (Chatterjee 1990; Tenenhouse 1990; Villamor et al. 2002). Vitamin D does not affect the process of bone mineralization directly, but mediates it through intestinal absorption and the consequent concentrations of calcium and phosphate influencing osteoblastic formation of matrix osteoid (Root 1990). Deficiency of vitamin D thus adversely affects bone growth and mineralization, potentially leading to rickets and bone deformity (Tenenhouse 1990). Vitamin C is found to be involved in the metabolism of connective tissues associated with calcium and phosphorus during calcification (Chatterjee 1990). Studies among living populations also demonstrated a positive effect of vitamin A supplementation on growth (Coles et al. 2004; Villamor et al. 2002). For example, a study among Israeli-Bedouin children revealed that stunting at age 12–18 months was associated with vitamin A deficiency and could be prevented by an additional dietary intake of this vitamin (Coles et al. 2004).

Childhood growth depends on the presence of necessary minerals and micronutrients in the diet, including those of calcium, phosphate, and zinc. A combined dietary deficiency of calcium and phosphate, or an isolated deficiency of either, can lead to hypocalcemia, hypophosphatemia, and rickets (Root 1990). The study of bone deformity in two Indian villages demonstrated association of skeletal deformities with local diet low in calcium (low milk intake) and high in phosphate (high consumption of potato) (Khandare et al. 2005). The authors also pointed out that prevalence of bone deformity correlates with higher levels of fluoride in drinking water in one of the villages.

Among many other minerals, zinc is also known for its influence on growth. It is addressed as an important element of skeletal growth due to it mediating role in DNA, RNA, protein, and the so-called growth factor synthesis, mineral metabolism, and bone remodeling (Wallwork and Sandstead 1990). For example, a study of the influence of zinc supplementation on height performed among undernourished growth-retarded Vietnamese children aged 4 to 36 months demonstrated an increase in growth velocity after zinc supplementation (Ninh et al. 1996).

Additionally, poor nutrition negatively affects immune responses and leads to depressed resistance to diseases (Eveleth and Tanner 1990). Protein deficiency and specific micronutrient deficiencies, including those of vitamins A and C, pyridoxine, folic acid, zinc and iron, adversely affect immune response (Dallman 1987; Scrimshaw et al. 1968). On this basis, various nutritional deficiencies are often synergistic with infectious diseases, which, in turn, negatively affect growth (Scrimshaw et al. 1968).

Role of Infectious Diseases. Infectious diseases adversely affect the process of growth. Infections can influence growth directly or indirectly, through negative effects on the nutritional status of the individual (Eveleth and Tanner 1990). Pathogens depress growth by interfering with the metabolism of essential elements for skeletal growth such as calcium and phosphorus, altering the set of amino acids, and reducing chondroblastic and osteoblastic activity (Scrimshaw et al. 1968). The direct effect of disease on growth was demonstrated in a survey of 650 children: those who did not have illnesses were one inch taller than their peers who had a high rate of infectious diseases, and 0.4 inches taller than those who were moderately ill (Scrimshaw et al. 1968).

Different infectious diseases of viral, bacterial, protozoal, and helminthes origin may affect normal growth. Numerous studies demonstrated a decrease in growth with increased levels of respiratory infections (Adair and Guilkey 1997; Hadi et al. 1999; Villamor et al. 2002). For example, Hadi and colleagues (1999) found that children free of respiratory infections significantly increased in height after supplementation of vitamin A, while those with a respiratory diseases load of >21.5% days during the period of observation did not respond to supplementation significantly.

Reduction of body height-to-age was found due to intestinal infections. A multivariate analysis of data collected among 3,000 Filipino children from birth to 24 months of age revealed that diarrhea was significantly correlated as one of the most likely reasons for stunting for 69% of rural children and 60% of urban children (Adair and Guilkey 1997). Another study among Burmese children aged from 2 to 12 years showed

that *Ascaris lumbricoides* infection was responsible for malnutrition and, consequently, low height-to-age and weight-for-age measurements, both of which were successfully improved after chemotherapeutic treatment of infection (Thein-Hlaing et al. 1991). The authors pointed out that severity of ascaric infection correlated with degree of malnutrition.

The interaction of two factors, infectious diseases and malnutrition, influences child growth to an important extent. Diseases adversely affect nutritional status by altering absorption, metabolism, and excretion of nutrients (Scrimshaw et al. 1968). In addition, food intake during the illness is usually reduced due to a negative effect of diseases on appetite and, in some cultures, traditional custom to limit the diet of the sick person (Chen 1979). Demands of the body for more protein needed for immune response to infectious disease might negatively influence growth, especially when the diet is insufficient in protein (Scrimshaw et al. 1968). Mata and colleagues (1977) in their study of Guatemalan children emphasized infectious diseases as the most important cause of the arrested growth and impaired physical development that commonly precipitated severe malnutrition and death, especially among children of weaning age (6 to 24 months). It was also noted that infectious diseases often predated kwashiorkor in malnourished children (Chen 1979). Many studies among living populations, however, indicate both malnutrition and infections as main factors that influence child growth, which emphasizes the importance of interaction between these factors (Crook 1994; Foster et al. 2005).

Studies on Archaeological Populations

Malnutrition. Reduction in stature was observed among many populations throughout the world with the adoption of agriculture (Cohen and Armelagos 1984). The researchers explained this fact by the concomitant heavy reliance on a single plant product and the subsequent decline in quality of diet, one lacking in necessary micronutrients and adequate amounts of animal protein. For example, Cook (1984), examining prehistoric juveniles from central Illinois, observed a decline in femoral length during the Late Woodland period compared to Middle Woodland. The author explains this fact as resulting from the adoption of a maize diet low in protein and iron and the limitation of other food resources in the Late Woodland period.

Diseases. Some anthropological studies emphasized the importance of disease load on child growth and resultant adult stature (Maat 2005; Mensforth 1985). Mensforth (1985) examined juvenile growth based on skeletal samples from Late Archaic (2655–3922 BC, Kentucky) and Late Woodland (AD 800–1100, Ohio) periods. The author found that the Late Woodland subadults showed a retarded growth rate compared to the Archaic sample. Mensforth argued that both populations had adequate nutrition. Based on observation of higher frequency of periosteal lesions (bone reaction to infections) in the Woodland sample, the author suggested that higher levels of infectious diseases were responsible for inhibited growth among subadults during the Late Woodland period.

Maat (2005) studied development of average male stature and it relationship with health in the Low Countries from AD 50 to the 20th Century. The results of the study

showed that there was a continuous decline in stature starting from 175.9 cm in the Roman period to 166 cm in the 17^{th}–18^{th} centuries, which was then followed by a gradual increase from the second half of the 19^{th} Century onward, reaching the present maximum of 184 cm. Analyzing the period of "negative secular trend," the author found that decline in stature correlated with high frequency of a skeletal marker of non-specific infections (11% tibial periostitis). Disease load due to increasing overcrowding of the cities, which led to poor hygiene and water contamination was among the main factors that influenced decrease of stature in the Low Countries.

Synergetic Influence of Malnutrition and Infectious Diseases. Most of the anthropological studies tend to see synergetic influence of dietary and infectious disease stresses on childhood growth and adult stature. For example, the study of egalitarian Yangshao culture and a chiefdom-like society of Longshan from Neolithic China (7000–4000 BP) shows decline of stature in the latter period (Pechenkina et al. 2002). The authors relate the decline in stature to a subsistence practice that was heavily dependent on millet as a staple and thus was insufficient in protein and micronutrients. It was pointed out that, although millet is naturally high in iron, as are many other types of cereal, it loses iron during the cooking process. The Longshan period is also characterized by population growth and enlargement of settlements, which provoked an increase in unsanitary conditions and consequent spread of infectious diseases. The researchers demonstrate that shorter stature occurred along with higher frequencies of such markers of physiological stress as linear enamel hypoplasia (indicator of non-

specific physiological stress), porotic hyperostosis, and cribra orbitalia (indicators of anemia). Thus, the authors conclude that decrease in stature in the Longshan period resulted from a combination of poor diet and increase in disease load (Pechenkina et al. 2002).

In addition to the adoption of agriculture, a decline in stature has been traced in archaeological contexts associated with other shifts of subsistence pattern. Lambert (1993) examined the change in stature in archaeological samples of native inhabitants of Santa Cruz and Santa Rosa islands over a period from 6000 BC to European contact. The researcher calculated a decrease in stature of about 10 cm. Analyzing associated archaeological materials, Lambert found that the islands inhabitants' diet gradually changed from a mixed hunter-gathering diet to one emphasizing protein-rich marine products. At the same time, islanders traded manufactured goods with mainland people to obtain seeds and roots. This fact allowed the author to suggest that islands' plant resources were inadequate and, consequently, the islanders' diet was lacking important nutrients. Lambert (1993) also found an increase in frequency of periosteal lesions (bone reaction to infections) along with an increase of sedentism and population density on the islands. The researcher concluded that decrease in stature resulted from increase in disease load and lack of necessary micronutrients in the diet.

Sexual Dimorphism. Sexual dimorphism in stature is another valuable parameter for evaluating communal health. In the studies on sexual dimorphism researchers recognize biological and environmental influences (Holden and Mace 1999; Stinson

1985). Generally, males have greater stature than females. Still, there is a significant variation in degree of sexual dimorphism between populations, and within one population, in different time periods. Data on sex differences in morbidity and growth often show that biologically males are less buffered against adverse environmental influences than females, but results are inconsistent (Stinson 1985).

Holden and Mace (1999) relate sexual dimorphism to different levels of female contribution to subsistence of the society. The authors found that in societies where women contribute a lot, they are taller, which leads to less sexual dimorphism and vice versa. The reason for this phenomenon, in the authors' opinion, is that sexual division of labor influences differential treatment that boys and girls receive during their growth. In other words, in societies where males contribute more to subsistence, parents would invest more in sons, providing for them better nutrition and health care (Holden and Mace 1999).

The studies of archaeological populations demonstrate variation in sexual dimorphism with social complexity. For example, low sexual dimorphism among an egalitarian society of the Badarian period in Ancient Egypt was explained by equal access of all people to food and other necessary resources, while increase in sexual dimorphism in the socially stratified Late Predynastic period was associated with different access to resources (Zakrzewski 2003). The author notes a slight increase in sexual dimorphism with time, arguing that it might reflect high status of women in ancient Egyptian society compared to women's status in other socially complex ancient populations. Tracing the change in sexual dimorphism, Zakrzewski (2003) observed that

the range of change in height through time was greater in males. The researcher suggests two causes to explain this finding: 1) females, being better buffered from adverse environmental factors, gained maximum genetically determined height in the early period or 2) with the development of social stratification, males received preferential access to food and medical care. Zakrzewski speculates that both scenarios might play a role in shaping sexual dimorphism in ancient Egypt (Zakrzewski 2003). Thus, shifting patterns of sexual dimorphism in stature can be possibly influenced, first, by biological differences in sensitivity of males and females to adverse environmental conditions and, second, by male and female differential disease load and access to food due to socioeconomic factors.

Correlation with Longevity. Adverse environmental factors that influence an individual's health during the period of growth and development lead to long-term consequences. Several studies show that an individual's height positively correlates with longevity. Jousilahti and colleagues (2000) surveyed around 32 thousand men and women living in eastern Finland and found that individual height has an inverse relationship with most of the measured risk parameters, including cardiovascular diseases, chronic pulmonary diseases, and total mortality. Subsequent studies on archaeological material answered the question whether this correlation existed in past centuries under probable different disease conditions. Investigations by Gunnel and colleagues (2001) and Kemkes-Grottenthaler (2005) have confirmed that shorter individuals died at a younger age. Kemkes-Grottenthaler (2005) calculated that survival

beyond age 40 improved by 15–16% for 1 SD of measurements of long bones. Thus, environmental influences that occurred during the growth period not only determine an individual height, but also affect an individual's health later in life.

Summary

Individual stature depends on many genetic and environmental factors. Genetic "programming" of potential height, biological schedule of growth spurts, and sex differences in sensitivity to environmental factors constitute significant components of resultant stature. Starting from the fetal stage of development, an individual undergoes various environmental influences, of which nutrition and disease load are the most important. Thus, to reach one's genetically predetermined stature, an individual has to have proper nutrition and healthy living conditions during the period of childhood growth. Inadequate nutrition and bad health history during the years of development not only lead to stunting but also influence adult health and longevity. The data on shifting patterns of sexual dimorphism in stature help to interpret adult stature as relating to differential treatment that both sexes receive in childhood and greater male vulnerability to environmental stresses. In order to understand population health status from stature measurements, we must take into consideration several different aspects, including subadult growth profile, ultimate adult stature, possible differences between social groups, sexual dimorphism in stature, and correlation of stature and longevity.

CHAPTER 3: METHODS

Enamel Hypoplasia

The sample for this study included both adult and subadult individuals with observable permanent dentition. Severe attrition is a known characteristic of this population (Jurmain 1990). To control for attrition, a mean value of crown height was calculated for each tooth type, based on data collected in my pilot study. Permanent teeth with degree of attrition more than 50% were excluded from the analysis. Teeth covered with calculus were also excluded. From approximately 300 individuals curated at San Jose State University, 85 with permanent dentition were included in the sample.

Three types of teeth (maxillary incisor, mandibular canine, and third mandibular premolar) were chosen for analysis of prevalence of enamel hypoplasia. Maxillary incisors and mandibular canines are considered to be the best indicators due to their high susceptibility to physiological stresses (Goodman and Armaelagos 1985). Posterior dentition is less hypoplastic and, thus, enamel hypoplasias on posterior dentition represent cases of more severe physiological stress. Dental anthropologists suggest different time standards for tooth development. According to Reid and Dean (2000), the observable labial enamel surface of the maxillary incisor reflects the record of amelogenesis during the span from 1.1 to 5 years, while observable enamel surface of mandibular canines represent the age from 1.5 to 6.2 years. The crown development of mandibular premolars continues from approximately 1.5–2 years of age to 5.5 years

(Smith 1991). Thus, the chosen types of teeth reflect the period of individual development from approximately 1 to 6 years of age. To avoid double counting, enamel hypoplasia was recorded on the teeth from the right side of the dentition; left side dentition was used in the case of tooth loss on the observed side. Teeth were examined under oblique light. A wooden probe was used to confirm of the depression on the enamel surface.

Twelve anterior permanent teeth of three types (central and lateral incisors and canines) were observed for presence or absence of enamel hypoplasia and further examined for number and position of hypoplastic defects on the crown. A subsample of these teeth, with no attrition, was used for analysis of distribution of hypoplastic lines by the time of occurrence. Studies on timing of tooth formation resulted in development of various charts and regression formulae for determination of chronological age at occurrence of hypoplastic defects. Goodman and Song (1999) demonstrated that choice of developmental standard, differences in tooth size among various populations, and consideration of time for formation of cuspal enamel are three main sources of variation and inaccuracy in estimation of age at occurrence. Although estimation of age-at-occurrence of hypoplasias is considered to be imprecise, it is still appropriate to analyze chronological distribution of enamel defects for comparative purposes within one population (Bartelink 2006). Reid and Dean's (2000) chart of enamel development was used in this study. This diagram has been chosen because it takes into account the length of time for formation of appositional enamel hidden beneath the cusp. The methodology applied in the Reid and Dean (2000) study made it possible to demonstrate that enamel

formation occurs with varying rather than with constant rate. It is recognized, however, that the chart was created based on a small sample and on a population not closely related to Native Americans, certainly leading to the potential for bias.

The position of the defect was measured following established procedure with fine-tipped calipers from CEJ (cemento-enamel junction) to the center of the defect in the case of thin lines (pits) and to the upper and lower borders of the defects in the case of extensive area of the crown involvement (Goodman et al. 1980; Malville 1997). The measurement of distance to hypoplastic defects was then converted to chronological age at occurrence.

Goodman and Song (1999) demonstrated in their study that the mean value of tooth height calculated for a particular population should be used for conversion estimations. I conducted a pilot study to determine a mean value of the crown height. Crown height was measured for all six types of anterior teeth: maxillary central incisors, maxillary lateral incisors, maxillary canines, mandibular central incisors, mandibular lateral incisors, and mandibular canines. The mean value of height was calculated for each type of tooth (Table 3.1).

Table 3.1. Mean Height of Maxillary and Mandibular Central and Lateral Incisors, and Canines.

MAXILLA	(in mm)	MANDIBLE	(in mm)
I1	11.7	I1	9.5
I2	9.9	I2	9.7
C	11.1	**C**	11.9

The number of stress episodes per individual was recorded by determining the number of hypoplastic defects matched by age. Thus, defects that did not have chronological matches will be excluded from the analysis of systematic stresses because of the possibility they could have formed due to local trauma or inflammation (Von Arx 1993).

Porotic Hyperostosis

All available crania were observed for presence or absence of porotic hyperostosis and cribra orbitalia. To retain consistency in recording data, I controlled for degree of preservation. For observation of porotic hyperostosis, only crania that have more than 50% of the frontal, parietals, and occipital bones adjacent to the lambdoidal, sagittal, and coronal sutures preserved were included in the sample. For observation of cribra

orbitalia, only orbits that have an orbital roof mostly preserved were included in the sample. If both orbits were present, the side with more pronounced expression was recorded. As a result, the samples for each variable comprised 196 individuals.

Both porotic hyperostosis and cribra orbitalia were recorded separately to trace the prevalence of each lesion in the sample. The degree of severity and status of healing were evaluated following the scoring system from *Standards for Data Collection* (Buikstra and Ubelaker 1994) and descriptive suggestions from Stuart-Macadam's study (1985): 1. indistinct porosity (very fine foramina), 2. slight or true porosity (scattered fine foramina), 3. medium (coalescent pores; large and small foramina that have linked to form trabeculae), and 4. severe (outgrowth of trabeculae from the contour of the cortical bone). Indistinct porosity was not included in the sample of affected individuals.

Periostitis

The sample was examined macroscopically for the presence of remodeling. The lesion was recorded for tibiae and the rest of the long bones separately according to data collection suggested by Steckel and colleagues (2002). The severity of involvement was scored as: 1. slight, when small patches of periosteal reaction cover less than one quarter of the bone; 2. moderate, when periostitis covers less than half of the bone; and 3. severe, when reaction involves more than half of the bone, including cases of osteomyelitis. The stage of healing was coded as: 1. active, when new, irregular, vascular bone is present; 2. healed, when remodeling is present in the form of mature lamellar bone; and 3. mixed,

when both types of bone remodeling are exhibited (Buikstra and Ubelaker 1994). The rest of the long bones were scored as: 1. periosteal lesion not related to trauma is present on any other long bone than the tibia; and 2. evidences of systemic infection throughout the skeleton, including the tibia (Steckel et al. 2002).

Prevalence of markers of infectious disease was calculated based on infectious lesions observed on tibiae. Of additional cases that lacked tibiae due to the state of preservation, only those that demonstrated evidence of severe systematic infections were included in a final count of frequency of infectious markers in the population under investigation. A total number of 217 individuals was included in the sample.

Stature

Reconstruction of stature from the length of long bones is an established procedure in physical anthropology. From the long bones, the femur shows the best correlation with stature (Feldesman 1992; Mendonca 2000). The sample included 134 adult individuals with at least one complete femur. The maximum length of the femur was measured with an osteometric board as it described in "Standards for Data Collection" (Buikstra and Ubelaker 1994). The measurements were recorded in millimeters. Following the suggestion of Steckel and colleagues (2002), the left femur was measured. The right femur was used in case the left was missing or damaged. In cases when adhesive was applied for femur reconstruction, approximate estimation was recorded.

Specific formulae are used for calculation of stature. The choice of the most appropriate formula presents an important issue. Because formulae are generated based on particular populations, any examined sample should be close to the reference population. This rule is especially strict when using regression formulae (Konigsberg et al. 1998). There are no formulae specifically created for any of the San Francisco Bay area prehistoric populations. Some of the researchers in the San Francisco Bay area used the Trotter and Gleser (1958) formula for Mongoloid populations on the basis of the relative genetic closeness of Native Americans to Asian populations (Grady et al. 2001). Scuilli and colleagues (1990) demonstrated, however, that this formula overestimates the stature of aboriginal population from the Ohio region by approximately 2 to 8 cm. No study has been conducted for appropriateness to use this formula for the bay area populations.

To avoid accumulation of errors related to calculation of stature, some of the authors used raw measurements of femora for direct comparison within the study (Kemkes-Grottenthaller 2005; Lambert 1993). Lambert (1993) also suggested that femur length, which accounts for approximately 27 percent of adult stature, can be used for rough estimation of stature. Following the suggestions, raw measurements of femur length were retained for comparison between subsamples within the present study.

Statistical Analysis

The population data were analyzed using the SPSS statistical package. The independent variables (factors) analyzed were sex, age, and affiliation to archaeological cultural strata of the Middle period, Late I period, and Late II period. Dependent variables were enamel hypoplasia, porotic hyperostosis (cranial and orbital lesions), markers of infectious diseases, and stature (i.e., femur length). An Analysis of Variance test (ANOVA) was considered to be the most appropriate method to analyze significant difference between means of dependent variables. For statistical analysis of association between variables, bivariate tests (Pearson correlation) were performed. The null hypothesis was chosen as representing a state where there is no significant difference between means of corresponding dependent variables for each independent variable. Significance levels were accepted when equal to or less than 0.05. Corresponding ANOVA values were calculated for every dependent variable and compared with accepted significance levels.

Variables Used in the Study

Six dependent variables were used in this study to assess health and its change over time in the examined archaeological population. The chosen dependent variables include following skeletal markers of general physiological stress: 1. enamel hypoplasia,

2. porotic hyperostosis, 3. cribra orbitalia, 4. periostitis, 5. stature (femoral length), 6. mortality (mean age at death). The three independent variables are: 1. temporal periods (Middle, Late I, and Late II), 2. sex (males, females, undetermined), and 3. age groups (subadults 0–15 and adults 16–55).

The four dependent variables, enamel hypoplasia, porotic hyperostosis, cribra orbitalia, and periostitis, were tested on differences by temporal periods, sex, and age groups. Because femoral length was examined for fully grown individuals (adults), this dependent variable was tested on differences by temporal period and sex. The dependent variable of mean age at death was examined for differences by temporal periods, sex, and association with other dependent variables. All dependent variables were examined for correlation with each other and combined effect on health.

CHAPTER 4: RESULTS

Enamel Hypoplasia

Tables 4.1 and 4.2 present the frequency of enamel hypoplasia by time periods and sex for each tooth type separately. Table 4.3 displays the frequency of enamel hypoplasia for adult and subadult age groups by time periods. Tables 4.4 through 4.6 show the results of statistical tests of a 3-way ANOVA run for three tooth types, revealing no significant correlation between prevalence of enamel hypoplasia by time periods, sex, and age subgroups.

Table 4.1. Frequency of Enamel Hypoplasia by Time Periods

Tooth type	Middle Period		Late I Period		Late II Period		Combined	
	N	%	N	%	N	%	N	%
Incisors	5/21	23.8	4/28	14.2	3/19	15.8	12/68	17.6
Canines	8/22	36.4	16/38	42.1	7/24	29.1	31/84	36.9
Premolars	4/19	21.0	6/30	20.0	3/19	15.8	13/68	19.1

Table 4.2. Frequency of Enamel Hypoplasia by Time Periods and Sex

	Middle Period				Late I				Late II			
	Males		Females		Males		Females		Males		Females	
	N	%	N	%	N	%	N	%	N	%	N	%
Incisors	2/9	22.2	2/10	20.0	1/11	9.1	3/16	18.7	3/15	20.0	0/2	0
Canines	3/9	33.3	3/11	27.2	8/17	47.1	7/19	36.8	6/20	30.0	0/2	0
Premolars	1/9	11.1	2/8	25.0	2/14	14.3	4/15	26.7	3/16	18.8	0/1	0

Table 4.3. Frequency of Enamel Hypoplasia by Age Groups

	Middle				Late I				Late II			
	0-15		16-55		0-15		16-55		0-15		16-55	
	N	%	N	%	N	%	N	%	N	%	N	%
Incisors	2/4	50.0	3/17	17.6	0/4	0.0	4/23	17.4	0/2	0.0	3/17	17.6
Canines	4/5	80.0	4/17	23.5	2/5	40.0	13/31	41.9	1/2	50.0	6/22	27.3
Premolars	2/5	40.0	2/14	14.3	0/4	0.0	6/24	25.0	0/2	0.0	3/17	17.6

Table 4.4. 3-way ANOVA Results for Maxillary Incisors

	Source	Sum of Squares	Df	F
Main effects	Combined	.266	5	.219
	Time periods	.217	2	.466
	Sex	.040	2	.082
	Age groups	.000	1	.000
Model		.266	5	.219
Residual		14.808	61	
Total		15.075	66	

Table 4.5. 3-way ANOVA Results for Mandibular Canines

	Source	Sum of Squares	Df	F
Main effects	Combined	2.826	5	.703
	Time periods	1.690	2	1.051
	Sex	.964	2	.600
	Age groups	.274	1	.341
Model		2.826	5	.703
Residual		61.076	76	
Total		63.902	81	

Table 4.6. 3-way ANOVA Results for Mandibular Premolar

	Source	Sum of Squares	Df	F
Main effects	Combined	.301	5	.236
	Time periods	.108	2	.211
	Sex	.079	2	.156
	Age groups	.047	1	.183
Model		.301	5	.236
Residual		15.290	60	
Total		15.591	65	

Because mandibular canines have the largest available sample size and are considered to be the most hypoplastic type of tooth, in depth analysis of adult and subadult differences in prevalence of hypoplasia between time periods was performed for this dental subsample. One-way ANOVA results showed that subadult had higher prevalence of hypoplastic defects in the Middle period (Table 4.7). The adult sample demonstrated increase in frequency between the Middle and the Late I periods (Table 4.8).

Table 4.7. One-way ANOVA Results for Adults/Subadults Differences in Prevalence of Hypoplasia by Time Periods (Canines)

	Source	Sum of Squares	Df	F
Middle	Between group	2.401	1	3.218 †
	Within group	14.918	20	
	Total	17.318	21	
Late I	Between group	.052	1	.056
	Within group	31.587	34	
	Total	31.639	35	
Late II	Between group	.015	1	.024
	Within group	13.818	22	
	Total	13.833	23	

†0.10<p<0.05

Table 4.8. ANOVA Results for Increase in Prevalence of Enamel Defects in Adults between the Middle and Late I Periods

	Source	Sum of Squares	Df	F
Mean age	Between sexes	13.451	1	2.789†
	Within sexes	607.728	126	
	Total	621.180	127	

†0.10<p<0.05

Table 4.9 shows the arithmetic means of mean age at death for enamel hypoplasia affected and defect-free individuals calculated separately for males and females by time periods. Table 4.10 presents the results of a 3-way ANOVA test for correlation between mean age at death with age, sex, and enamel defects.

Table 4.9. Mean Ages at Death for Individuals with and without Enamel Hypoplasias

Tooth type	Sex	Time period	With hypoplasia		Without hypoplasia		Difference (years)
			Mean	SD	Mean	SD	
Canines	Male	Middle	28.33	12.22	24.00	9.38	-4.33
		Late I	23.86	8.13	28.31	7.60	4.45
		Late II	29.33	7.55	30.21	6.70	0.88
		Combined	26.75	8.51	28.34	7.68	1.59
	Female	Middle	20.33	5.69	25.63	5.99	5.30
		Late I	21.00	3.74	29.00	9.97	8.00
		Late II	Nd	nd	27.50	2.12	nd
		Combined	20.80	4.08	27.57	8.07	6.77
	Total	Middle	24.33	9.58	24.93	7.34	0.60
		Late I	22.43	6.26	28.71	8.82	6.28
		Late II	29.33	7.55	29.86	6.34	0.53
		Combined	24.46	7.63	28.01	7.78	3.55

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Tooth type	Sex	Time period	With hypoplasia		Without hypoplasia		Difference (years)
			Mean	SD	Mean	SD	
moderate/severe vs. no defects	Male	Middle	35.00	5.66	21.60	8.17	-13.40
		Late I	18.67	1.53	28.31	7.60	9.64
		Late II	24.67	7.09	30.04	7.10	5.37
		Combined	25.00	8.09	27.80	7.85	2.80
	Female	Middle	18.00	5.66	25.71	6.47	7.71
		Late I	22.60	2.30	29.00	9.97	6.40
		Late II	Nd	nd	27.50	2.12	nd
		Combined	21.29	3.73	27.70	8.26	6.41
	Total	Middle	21.83	11.09	24.00	7.19	2.17
		Late I	21.13	2.80	28.71	8.82	7.58
		Late II	24.67	7.09	29.68	6.62	5.01
		Combined	22.00	7.06	27.76	7.94	5.76

Table 4.10. 3-way ANOVA Results for Correlation between Mean Age at Death, Sex, and Enamel Defects

	Source	Sum of Squares	Df	F
Main effects	Combined	1746.071	6	5.225***
	Time periods	172.956	2	1.553
	Sex	1180.394	2	10.596***
	Enamel hypoplasia	143.970	2	1.292
Model		1746.071	6	5.225***
Residual		4177.445	75	
Total		5923.515	81	

***p<0.000

Due to significant values for sex involvement found with the 3-way ANOVA test, the sample was further analyzed for difference in mean age by sex. The Late II period subsample was not included in the analyses due to lack of data for females. When individuals of undetermined sex were excluded from the analysis, no significant differences in mean age at death were found between sexes (Table 4.11).

Table 4.11. ANOVA Results for Difference in Mean Age at Death for Males and Females with Enamel Hypoplasia

	Source	Sum of Squares	Df	F
Mean age	Between sexes	137.500	1	2.725
	Within sexes	1009.273	20	
	Total	1146.773	21	

For further analyses of enamel hypoplasia stress only moderately to severely affected individuals, the ones who had two or three tooth types affected, were selected. Table 4.12 shows that moderately to severely affected individuals had shorter mean age at death than individuals who had no enamel defects.

Table 4.12. ANOVA Results for Difference in Mean Age at Death for Individuals with Moderate/Severe Hypoplasia and Hypoplasia-Free

	Source	Sum of Squares	Df	F
Mean age	Between groups	316.106	1	4.723*
	Within groups	4149.702	62	
	Total	4465.809	63	

*$p<0.05$

Enamel stresses were also analyzed by age at occurrence. Table 4.13 shows the mean age of hypoplasia formation by tooth type and time periods. Table 4.14 presents

mean age of defect formation by age groups, comparing those who died before age 20 with those who died after age 20. The ANOVA test confirmed that individuals who died before age 20 had earlier onset of hypoplastia than those who died after age 20 (Table 4.15).

Table 4.13. Mean Age of Enamel Defects Formation by Time Periods

	Incisors			Canines		
Period	Mean	N	SD	Mean	N	SD
Middle	3.34	13	.59	3.98	22	.48
Late I	3.37	22	.71	4.17	36	.53
Late II	3.17	10	.93	4.06	13	.58
Combined	3.31	45	.72	4.09	71	.53

Table 4.14. Mean Age of Enamel Defects Formation by Time Periods and Age Groups

Time Period	Incisors		Canines	
	< 20 yrs	> 20 yrs	< 20 yrs	> 20 yrs
Middle	3.26	3.66	3.95	4.08
Late I	3.13	3.78	3.99	4.42
Late II	3.17	Nd	4.05	4.12
Combined	3.19	3.76	3.99	4.31

Table 4.15. ANOVA Results for Mean Age of Enamel Defects Formation by Age Groups

	Source	Sum of Squares	df	F
Incisors	Between groups	2.536	1	5.305*
	Within groups	20.556	43	
	Total	23.092	44	
Canines	Between groups	1.557	1	5.917*
	Within groups	18.159	69	
	Total	19.716	70	

* $p<0.05$

Porotic Hyperostosis and Cribra Orbitalia

Cranial Lesions

Table 4.16 presents the frequency of porotic cranial lesions by time periods. Table 4.17 and Figure 4.1 show distribution of porotic lesions between sexes and time periods. Table 4.18 displays the frequency of porotic hyperostosis for adult and subadult age groups by time periods. Table 4.19 shows the results of statistical tests of 3-way ANOVA, which revealed a significant value for combined effects, and a marginally significant value for time periods.

Table 4.16. Frequency of Porotic Cranial Lesions by Time Periods and Degree of Severity

	Middle		Late I		Late II		Combined	
	N	%	N	%	N	%	N	%
Mild	17/44	38.6	52/99	52.5	27/53	50.9	96/196	49.0
Moderate	2/44	4.5	5/99	5.1	1/53	1.9	8/196	4.1
Severe	-	-	1/99	1.0	-	-	1/196	0.5
Total	19/44	43.2	58/99	58.6	28/53	52.8	105/196	53.6

Table 4.17. Frequency of Porotic Cranial Lesions by Sex and Time Periods

Time Period	Males		Females	
	N	%	N	%
Middle	12/19	63.1	7/17	41.2
Late I	24/31	77.4	26/41	63.4
Late II	14/25	56.0	10/15	66.7
Combined	50/75	66.7	43/73	58.8

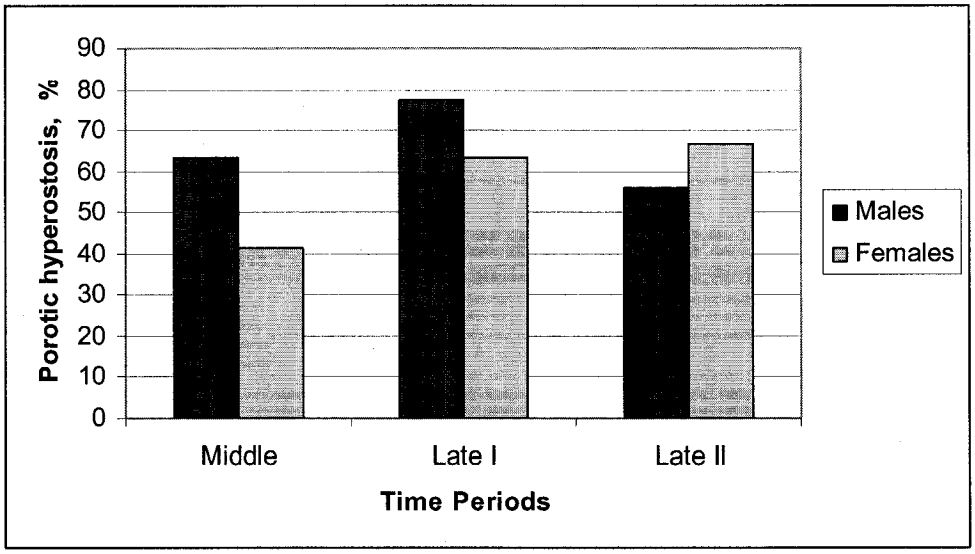

Figure 4.1. Distribution of porotic hyperostosis by sex and time periods

Table 4.18. Frequency of Porotic Lesions by Age Groups

Time period	Age group 0-15		Age group 16-55	
	N	%	N	%
Middle	0/11	0.0	19/33	57.6
Late I	10/29	34.5	45/67	67.1
Late II	3/11	27.3	24/40	60.0
Combined	13/51	25.5	88/140	62.9

Table 4.19. 3-way ANOVA Results for Porotic Hyperostosis

	Source	Sum of Squares	df	F
Main effects	Combined	12.965	6	3.010**
	Time periods	4.241	2	2.954†
	Sex	1.164	2	.810
	Age groups	1.241	2	.864
Model		12.965	6	3.010**
Residual		135.703	189	
Total		148.668	195	

†0.10<p<0.05, **p<0.01

Differences between time periods in porotic hyperostosis indicated by 3-way ANOVA were further examined for combined sexes and each sex separately. An ANOVA test revealed marginally significant increase in porotic hyperostosis between the Middle and Late I periods (Table 4.20). When the sample was controlled for sex, males demonstrated significance of decrease in porotic lesions between the Late I and Late II periods (Table 4.21).

Table 4.20. ANOVA Results for Increase in Porotic Hyperostosis between the Middle and Late I Periods

	Source	Sum of Squares	df	F
Porotic hyperostosis	Between time periods	2.455	1	2.978†
	Within time periods	115.411	140	
	Total	117.866	141	

†0.10<p<0.05

Table 4.21. ANOVA Results for Decrease in Porotic Hyperostosis among Males between the Late I and Late II Periods

	Source	Sum of Squares	df	F
Porotic hyperostosis	Between time periods	2.695	1	4.379*
	Within time periods	33.234	54	
	Total	35.929	55	

*p<0.05

Time period differences in porotic hyperostosis were further analyzed for adult and subadult age groups. One-way ANOVA tests demonstrated significantly higher

prevalence of cranial porotic lesions in the adult group in the Middle and Late II periods, but not in the Late I period (Table 4.22). An ANOVA test with the subadult group selected revealed a marginally significant increase in porotic hyperostosis in this age group between the Middle and Late I periods (Table 4.23). Examination of cranial lesions by state of healing showed that active lesions, which were observed only in subadults, increase in the Late I period (Table 4.24). This observation, however, was not possible to test statistically due to small sample size.

Table 4.22. ANOVA Results for Difference in Porotic Hyperostosis between Adults/Subadults by Time Periods

	Source	Sum of Squares	df	F
Middle	Between groups	5.523	1	9.312**
	Within groups	24.909	42	
	Total	30.432	43	
Late I	Between groups	1.679	1	1.903
	Within groups	82.047	93	
	Total	83.726	94	
Late II	Between groups	2.792	1	5.239*
	Within groups	26.650	50	
	Total	29.442	51	

*p<0.05, **p<0.01

Table 4.23. ANOVA Results for Increase in Porotic Hyperostosis between the Middle and Late I Periods for Subadults

	Source	Sum of Squares	df	F
Porotic hyperostosis	Between time periods	3.636	1	3.945†
	Within time periods	34.107	37	
	Total	37.744	38	

†0.10<p<0.05

Table 4.24. Distribution of State of Healing of Porotic Hyperostosis by Time Periods

Time period	Active		Healed /Healing	
	N	%	N	%
Middle	0/44	0	19/44	43.2
Late I	8/99	8.1	50/99	50.5
Late II	1/52	1.9	27/52	51.9
Combined	9/195	4.6	96/152	63.2

To assess effect of porotic hyperostosis on an individual's longevity, the arithmetic means of mean age at death for porotic hyperostosis affected and defect-free individuals were calculated separately for males and females by time periods (Table

4.25). Table 4.26 presents the results of a 3-way ANOVA test for correlation between mean age at death with age, sex, and porotic defects.

Table 4.25. Mean Age at Death of Individuals with and without Porotic Hyperostosis

Sex	Time period	Individuals with porotic hyperostosis		Individuals without porotic hyperostosis		Difference (in years)
		Mean	SD	Mean	SD	
Male	Middle	35.41	7.76	24.28	12.05	-11.13
	Late I	31.29	8.25	31.85	9.56	0.56
	Late II	30.07	6.97	32.73	8.03	2.66
	Combined	31.98	7.88	30.12	10.0	-1.86
Female	Middle	25.14	6.12	30.40	8.90	5.26
	Late I	31.33	9.63	35.07	8.44	3.74
	Late II	38.22	7.61	37.20	6.61	-1.02
	Combined	31.80	9.48	33.87	8.48	2.07
Total	Middle	31.63	8.67	27.88	10.43	-3.75
	Late I	31.31	8.91	34.05	8.71	2.74
	Late II	33.26	8.14	34.13	7.70	0.87
	Combined	31.90	8.60	32.16	9.30	0.26

Table 4.26. 3-way ANOVA Results for Correlation between Porotic Hyperostosis, Mean Age at Death, and Sex

	Source	Sum of Squares	Df	F
Main effects	Combined	16188.503	5	45.773***
	Time periods	264.580	2	1.870
	Sex	10254.904	2	72.490***
	Porotic hyperostosis	312.547	1	4.419*
Model		26518.469	13	28.839***
Residual		12378.318	175	
Total		38896.787	188	

*p<0.05, ***p<0.001

Significant values for sex differences displayed by the 3-way ANOVA test led to further examination of sex differences in mean age at death of individuals affected by porotic hyperostosis. A post-hoc Bonferroni test revealed significant differences in mean age only between males/females and the undetermined sex category, most of which was comprised of subadults (Table 4.27). When all sexes were included, moderately to severely affected individuals showed shorter life span than non-affected (Table 4.28).

Table 4.27. Post-Hoc Results for Sex Differences in Mean Age at Death for Individuals Affected with Porotic Hyperostosis

Sex	Source	Mean Difference	Standard Error
Males	Females	.1787	1.9914
	Undetermined	22.0204*	2.9941
Females	Males	-.1787	1.9914
	Undetermined	21.8417*	3.0470
Undetermined	Males	-22.0204*	2.9941
	Females	-21.8417*	3.0470

*p<0.05

Table 4.28. ANOVA Results for Lower Mean Age at Death of Individuals with Moderate/Severe Porotic Lesions vs. Non-Affected Individuals

	Source	Sum of Squares	Df	F
Porotic hyperostosis	Between groups	2318.605	3	3.518*
	Within groups	26584.811	121	
	Total	28903.416	124	

*p<0.05

Significant difference in mean age at death between affected and non-affected individuals found by a 3-way ANOVA test was not confirmed when subadults were excluded from the analysis (Table. 4.29).

Table 4.29. ANOVA Results for Difference in Mean Age at Death of Adult Individuals Affected with Porotic Hyperostosis vs. Non-Affected Individuals

	Source	Sum of Squares	Df	F
Porotic hyperostosis	Between time periods	367.313	3	1.582
	Within time periods	10677.187	138	
	Total	11044.500	141	

Analysis of the sample, while controlling for sex, showed that females had a significantly higher mean age at death than affected males in the Late II period (Table 4.30).

Table 4.30. ANOVA Results for Sex Differences in Mean Age at Death for Porotic-Affected Individuals (Late II Period)

	Source	Sum of Squares	Df	F
Porotic hyperostosis	Between males and females	363.951	1	6.983*
	Within males and females	1094.484	21	
	Total	1458.435	22	

*$p<0.05$

Orbital Lesions

Table 4.31 presents the frequency of cribra orbitalia by time periods. Table 4.32 and Figure 4.2 show the distribution of orbital lesions between sexes and time periods. Table 4.33 displays the frequency of cribra orbitalia for adult and subadult age groups by time periods. Table 4.34 shows the results of statistical tests of 3-way ANOVA for cribra orbitalia, revealing significant value for combined effects and a marginally significant value for sex differences.

Table 4.31. Frequency of Orbital Lesions by Time Period and Degree of Severity

	Middle		Late I		Late II		Combined	
	N	%	N	%	N	%	N	%
Mild	7/44	15.9	28/99	28.3	9/53	17.0	44/196	22.4
Moderate	0/44	0	3/99	3.0	2/53	3.8	5/196	2.6
Severe	1/44	2.3	1/99	1.0	0/53	0	2/196	1.0
Totally	8/44	18.2	32/99	32.3	11/53	20.8	51/196	26.0

Table 4.32. Frequency of Orbital Lesions by Sex and Time Period

Time Period	Males		Females	
	N	%	N	%
Middle	3/20	15.0	3/16	18.8
Late I	6/31	19.4	15/40	37.5
Late II	2/23	8.7	5/13	38.5
Combined	11/74	14.9	23/69	33.3

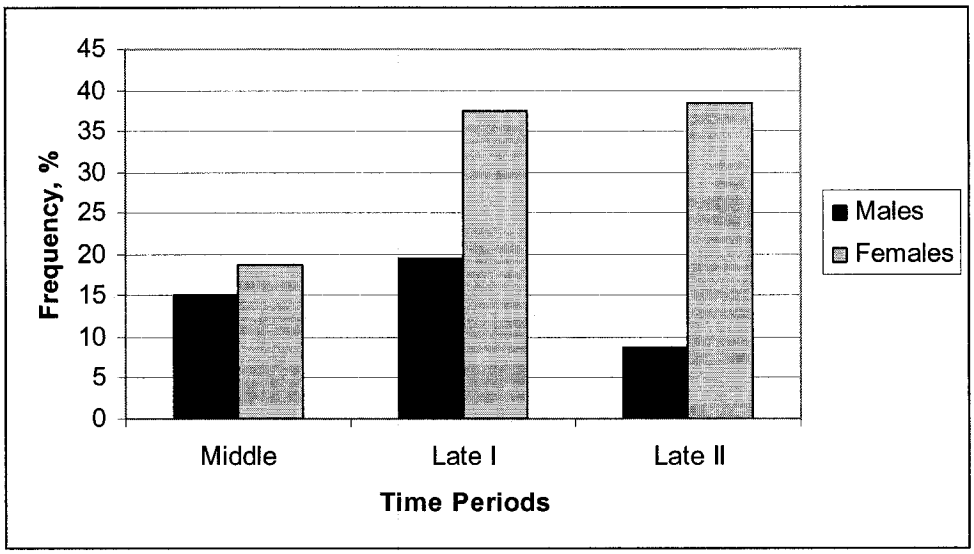

Figure 4.2. Distribution of cribra orbitalia by sex and time periods

Table 4.33. Distribution of Porotic Lesions between Adults and Subadults

Time period	Age group 0-15		Age group 16-55	
	N	%	N	%
Middle	4/11	36.4	4/33	12.1
Late I	11/30	36.7	21/66	31.8
Late II	4/15	26.7	7/36	19.4
Combined	19/56	33.9	32/135	23.7

Table 4.34. 3-way ANOVA Results for Cribra Orbitalia

	Source	Sum of Squares	df	F
Main effects	Combined	2.589	6	2.321*
	Time periods	.652	2	1.753
	Sex	.942	2	2.534†
	Age groups	.491	2	1.319
Model		2.589	6	2.321*
Residual		35.141	189	
Total		37.730	195	

†0.10<p<0.05, *p<0.05

Due to a marginally significant value for sex differences demonstrated by the 3-way ANOVA test (F = 2.534, df = 2), orbital lesions were further examined for each sex separately (Table 4.35). Analysis showed that, compared to males, females were more affected with cribra orbitalia, especially in the Late II period (Figure 4.2 and Table 4.36).

Table 4.35. Post-Hoc Results for Sex Differences in Cribra Orbitalia

Sex	Source	Mean Difference	Standard Error
Males	Females	-.185*	.073
	Undetermined	-.172	.078
Females	Males	.185*	.073
	Undetermined	.013	.079
Undetermined	Males	.172	.078
	Females	-.013	.079

*p<0.05

Table 4.36. ANOVA Results for Males/Females Difference in Cribra Orbitalia

	Source	Sum of Squares	df	F
Middle	Between sexes	.013	1	.085
	Within sexes	4.988	34	
	Total	5.000	35	
Late I	Between sexes	.575	1	2.791†
	Within sexes	14.214	69	
	Total	14.789	70	
Late II	Between sexes	.736	1	5.103*
	Within sexes	4.903	34	
	Total	5.639	35	
Combined	Between sexes	1.218	1	6.953**
	Within sexes	24.698	141	
	Total	25.916	142	

†0.10<p<0.05, *p<0.05, **p<0.01

Table 4.37 presents the distribution of cribra orbitalia by state of healing. Active lesions, which were observed only in the subadult group, showed a marginally significant increase in the Late I period (Table 4.38). Healed and healing lesions were observed mostly in the adult group, which demonstrated increase in prevalence and severity of cribratic lesions between the Middle and Late I periods (Table 4.39).

Table 4.37. Distribution of Orbital Lesions by State of Healing

State of healing	Middle		Late I		Late II		Combined Periods	
	N	%	N	%	N	%	N	%
Active	1/46	2.2	10/99	10.1	4/53	7.5	15/51	29.4
Healing/Healed	7/46	15.2	22/99	22.2	5/53	9.4	36/51	70.6

Table 4.38. ANOVA Results for Increase in Active Lesions of Cribra Orbitalia between the Middle and Late I Periods

	Source	Sum of Squares	df	F
Active Lesions	Between time periods	.272	1	3.127†
	Within time periods	9.657	111	
	Total	9.929	112	

†$0.10<p<0.05$

Table 4.39. ANOVA Results for Increase in Orbital Lesions between the Middle and Late I Periods (Adults)

	Source	Sum of Squares	df	F
Active Lesions	Between time periods	.854	1	4.643*
	Within time periods	17.833	97	
	Total	18.687	98	

*p<0.05

Table 4.40 presents the arithmetic means of ages at death calculated for cribra orbitalia affected and non-affected individuals in order to assess effect of anemia on longevity. Table 4.41 shows the results of a 3-way ANOVA test for correlation between mean age at death and temporal periods, sex, and cribra orbitalia.

Table 4.40. Mean Age at Death of Individuals with and without Cribra Orbitalia

Sex	Time period	Individuals with cribra orbitalia		Individuals without cribra orbitalia		Difference (years)
		Mean	SD	Mean	SD	
Male	Middle	27.67	12.70	32.35	10.45	4.68
	Late I	25.50	9.12	32.93	7.87	7.43
	Late II	24.50	10.60	32.43	6.93	7.93
	Combined	25.91	9.30	32.59	8.24	6.68
Female	Middle	26.67	13.58	28.85	7.43	2.18
	Late I	31.86	9.88	32.16	9.39	0.30
	Late II	35.60	6.19	38.71	8.20	3.11
	Combined	32.00	9.62	32.22	9.07	0.22
Total	Middle	27.17	11.77	30.83	9.28	3.66
	Late I	29.95	9.88	32.53	8.61	2.58
	Late II	32.43	8.58	34.00	7.63	1.57
	Combined	29.97	9.81	32.44	8.56	2.47

Table 4.41. 3-way ANOVA Results for Effects of Cribra Orbitalia on Mean Age at Death by Sex and Time Periods

	Source	Sum of Squares	df	F
Main effects	Combined	14523.993	5	38.267***
	Time periods	94.630	2	.623
	Sex	13662.276	2	89.991***
	Cribra orbitalia	209.572	1	2.761†
Model		27891.019	17	21.613***
Residual		12904.518	170	
Total		40795.536	187	

†0.10<p<0.05, ***p<0.001

Significant value for sex differences (F = 89.991, df = 2) analyzed with Post-hoc Bonferroni test revealed that mean age at death was lower for individuals of undetermined sex (mostly subadults) but not significantly different between males and females (Table 4.42). Affected females demonstrated slightly higher mean age at death than affected males (Table 4.43). When the sample was controlled for sex, affected males on average showed lower mean age at death compared to non-affected males (Table 4.44). Also, affected individuals of all sexes showed lower mean age at death compared to non-affected individuals (Table 4.45).

Table 4.42. Post-Hoc Results for Sex Differences in Mean Age at Death for the Cribra Orbitalia Sample

Sex	Source	Mean Difference	Standard Error
Males	Females	-1.6618	1.3567
	Undetermined	24.7866*	1.3416
Females	Males	1.6618	1.3567
	Undetermined	26.4484*	1.3637
Undetermined	Males	-24.7866*	1.3416
	Females	-26.4484*	1.3637

*p<0.05

Table 4.43. ANOVA Results for Difference in Mean Age at Death between Cribra Orbitalia Affected Males vs. Affected Females

	Source	Sum of Squares	df	F
Mean age	Between time periods	272.061	1	3.007†
	Within time periods	2804.909	31	
	Total	3076.970	32	

†0.10<p<0.05

Table 4.44. ANOVA Results for Difference in Mean Age at Death for Males Affected with Cribra Orbitalia vs. Non-Affected

	Source	Sum of Squares	df	F
Mean age	Between time periods	415.120	1	5.876*
	Within time periods	4874.655	69	
	Total	5289.775	70	

*p<0.05

Table 4.45. ANOVA Results for Difference in Mean Age at Death for Individuals Affected with Cribra Orbitalia vs. Non-Affected

	Source	Sum of Squares	df	F
Mean age	Between time periods	720.233	1	3.343†
	Within time periods	40075.303	186	
	Total	40795.536	187	

†0.10<p<0.05

Periostitis

Table 4.46 presents the frequency of periosteal lesions by time periods and degree of severity. Table 4.47 and Figure 4.3 show distribution of periostitis between sexes and time periods. Table 4.48 and Figure 4.4 display the frequency of periostitis for adult and subadult age groups by time periods. Table 4.49 shows the results of a 3-way ANOVA test, which revealed only marginally significant values for differences among sex and age groups.

Table 4.46. Frequency of Periostitis by Time Period and Severity

	Middle		Late I		Late II		Combined	
	N	%	N	%	N	%	N	%
All levels	9/47	19.1	21/101	20.8	19/69	27.5	49/217	22.6
Moderate/severe	3/47	6.4	6/101	5.9	9/69	13.0	18/217	8.3

Table 4.47. Frequency of Periostitis for Males and Females by Time Period

	Middle		Late I		Late II		Combined	
	N	%	N	%	N	%	N	%
Males	3/20	15.0	7/34	20.6	6/29	20.7	16/83	19.3
Females	3/16	18.8	12/44	27.3	10/23	43.5	25/83	30.1

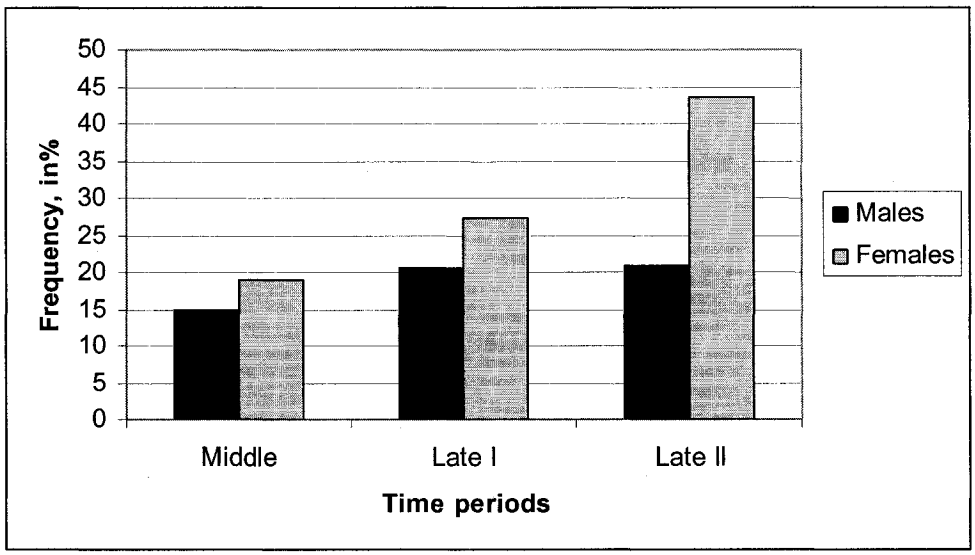

Figure 4.3. Frequency of periosteal lesions for males and females by time periods

Table 4.48. Distribution of Periostitis among Adults and Subadults by Time Period

		Middle		Late I		Late II		Combined	
		0-15	16-55	0-15	16-55	0-15	16-55	0-15	16-55
All levels	N	4/12	4/34	2/24	19/79	1/14	16/51	7/50	39/159
	%	33.3	11.8	8.3	24.1	7.1	31.4	14.0	24.5
Moderate/ Severe	N	1/12	1/34	2/24	4/79	0/14	9/51	3/50	14/159
	%	8.3	2.9	8.3	5.1	0	17.6	6.0	8.8

Table 4.49. 3-way ANOVA Results for Periostitis

	Source	Sum of Squares	Df	F
Main effects	Combined	1.194	5	1.431
	Time periods	.402	2	1.205
	Sex	.797	2	2.389†
	Age groups	.537	1	3.220†
Model		3.342	13	1.541
Residual		32.534	195	
Total		35.876	208	

†0.10<p<0.05

When the sample was controlled for sex, females from the Late II period demonstrated slightly higher frequency of lesions than males but this pattern was mostly due to prevalence of slight lesions (Table 4.50 and Figure 4.3). However, when periosteal lesions were tested by severity, males from the Late II period showed significantly higher prevalence of severe lesions compared to females (Table 4.51).

Table 4.50. ANOVA Results for Prevalence in Periostitis among Females in the Late II Period

	Source	Sum of Squares	df	F
Periostitis	Between sexes	.666	1	3.199†
	Within sexes	10.411	50	
	Total	11.077	51	

†0.10<p<0.05

Table 4.51. ANOVA Results for Prevalence of Severe Periosteal Lesions among Males in the Late II Period

	Source	Sum of Squares	Df	F
Periostitis	Between time periods	1.000	1	5.091*
	Within time periods	2.750	14	
	Total	3.750	15	

*p<0.05

Analysis of frequency of periostitis by age groups and time periods showed significant increase in prevalence of periosteal lesions in adults between the Middle and Late II periods (Table 4.52 and Figure 4.4). On the contrary, prevalence of periosteal

lesions in subadults slightly decreases between the Middle and Late II periods (Table 4.53).

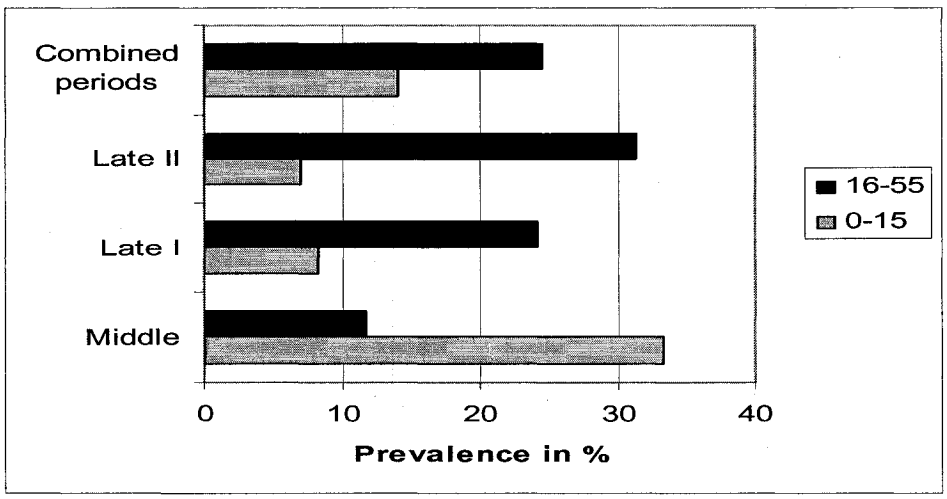

Figure 4.4. Distribution of frequency of periostitis by age groups and time periods

Table 4.52. ANOVA Results for Increase in Periostitis in Adult Group between the Middle and Late II Periods

	Source	Sum of Squares	Df	F
Periostitis	Between time periods	.784	1	4.486*
	Within time periods	14.510	83	
	Total	15.294	84	

*p<0.05

Table 4.53. ANOVA Results for Decrease in Periostitis in Subadult Group between the Middle and Late II Periods

	Source	Sum of Squares	Df	F
periostitis	Between time periods	.443	1	2.959†
	Within time periods	3.595	24	
	Total	4.038	25	

†0.10<p<0.05

To assess effect of periostitis on longevity, the arithmetic means of age at death were calculated for affected and non-affected individuals who had available data for age and sex (Table 4.54). Table 4.55 shows the results of 3-way ANOVA for correlations between mean age at death and temporal periods, sex, and periosteal markers.

Table 4.54. Mean Age at Death for Individuals with and without Periostitis

Sex	Time period	With periostitis		Without periostitis		Difference (years)
		Mean	SD	Mean	SD	
Male	Middle	37.5	3.5	32.1	10.3	-5.4
	Late I	35.6	9.2	30.1	7.9	-5.5
	Late II	26.1	10.0	34.0	7.2	7.9
	Combined	32.0	10.0	32.0	8.4	0.0
Female	Middle	24.7	10.5	29.3	8.0	4.6
	Late I	33.5	9.7	32.6	9.5	-0.9
	Late II	38.3	4.9	37.8	7.2	-0.5
	Combined	34.3	9.1	32.8	9.0	-1.5
Total	Middle	29.8	10.4	30.9	9.3	1.1
	Late I	34.3	9.3	31.5	8.9	-2.8
	Late II	33.5	9.4	35.0	7.3	1.5
	Combined	33.4	9.3	32.4	8.6	-1.0

Table 4.55. 3-way ANOVA Results for Mean Age at Death by Temporal Periods, Sexes, and Periostitis

	Source	Sum of Squares	Df	F
Main effects	Combined	13701.649	5	37.827***
	Time periods	326.055	2	2.250
	Sex	11302.505	2	78.008***
	Periostitis	12.161	1	.168
Model		28897.154	17	23.464***
Residual		13691.928	189	
Total		42589.082	206	

***p<0.001

Due to a significant value for sex differences revealed by 3-way ANOVA (F = 78.008, df = 2), sex differences in mean age at death were further analyzed. A post-hoc test showed a significantly different mean age at death only between males/females and individuals of undetermined sex, which was comprised mostly of subadults (Table 4.56).

Table 4.56. Post-Hoc Results for Sex Differences in Mean Age at Death for Individuals Affected with Periostitis

Sex	Source	Mean Difference	Standard Error
Males	Females	-1.2655	1.4083
	Undetermined	26.1521*	1.6210
Females	Males	1.2655	1.4083
	Undetermined	27.4176*	1.6249
Undetermined	Males	-26.1521*	1.6210
	Females	-27.4176*	1.6249

*$p<0.05$

When the sample was controlled for sex and time periods, an ANOVA test showed that, among individuals affected with periostitis, females had a higher mean age at death than males in the Late II period only (Table 4.57). Females, both affected and non-affected with periostitis, showed increase in mean age at death between the Middle and Late II periods (Table 4.58). In contrast, males affected with periostitis showed lower mean age at death than non-affected males in the Late II period (Table 4.59).

Table 4.57. ANOVA Results for Higher Mean Age at Death of Periostitis-Affected Females Compared To Periostitis-Affected Males (Late II Period)

	Source	Sum of Squares	Df	F
Mean age	Between males and females	550.069	1	10.357**
	Within males and females	690.431	13	
	Total	1240.500	14	

**p<0.01

Table 4.58. ANOVA Results for Mean Age at Death for Females with Periostitis (the Middle vs. Late II Periods)

	Source	Sum of Squares	Df	F
Affected females	Between periods	427.111	1	10.295**
	Within periods	414.889	10	
	Total	842.000	11	
Non-affected females	Between periods	369.231	1	6.353*
	Within periods	1336.769	23	
	Total	1706.000	24	

*p<0.05, **p<0.01

Table 4.59. ANOVA Results for Mean Age at Death for Males Affected with Periostitis vs. Non-Affected (Late II Period)

	Source	Sum of Squares	Df	F
Mean age	Between males and females	294.973	1	4.835*
	Within males and females	1647.165	27	
	Total	1942.138	28	

*$p<0.05$

Stature

Table 4.60 shows the arithmetic means for femoral length by sex and time periods. Figure 4.5 displays a graphic representation of changes in femoral length by time periods for each sex separately. Table 4.61 shows the results of the 2-way ANOVA test for femoral length with a significant value for combined effects and sex.

Table 4.60. Mean Femoral Length by Time Period and Sex

Time period	Sex	Mean (in mm)	N	SD
Middle	Male	442.3	15	14.4
	Female	411.3	12	12.3
	Total	429.0	28	22.8
Late I	Male	439.5	26	19.8
	Female	408.9	39	18.1
	Total	421.1	65	24.0
Late II	Male	434.2	24	17.7
	Female	414.1	17	22.1
	Total	425.9	41	21.8
Combined	Male	438.2	65	17.9
	Female	410.8	68	20.56
	Total	424.2	134	23.3

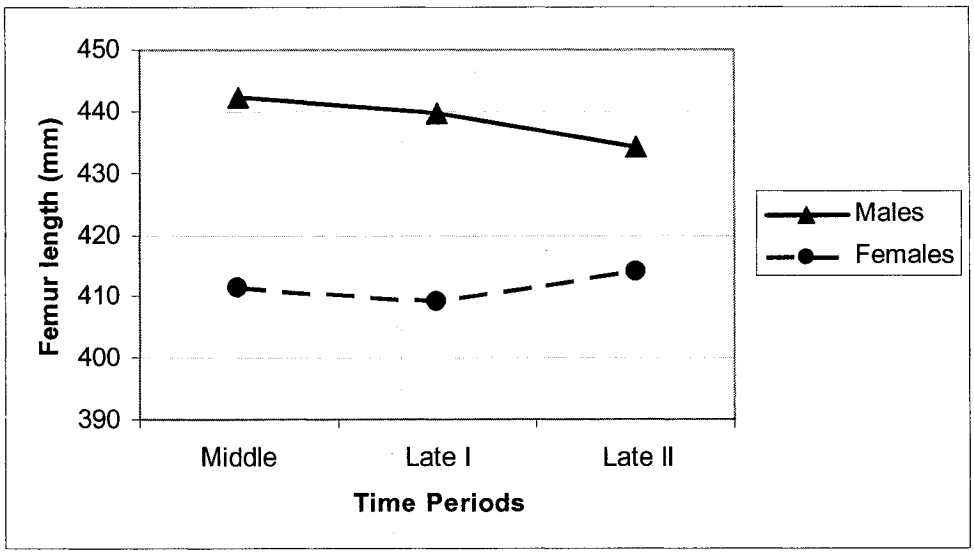

Figure 4.5. Males and females mean femoral length by time period

Table 4.61. 2-way ANOVA Results for Femur Length by Time Period and Sex

	Source	Sum of Squares	df	F
Main effects	Combined	22088.989	3	22.362***
	Time periods	145.521	2	.221
	Sex	21192.750	1	64.365***
2-Way Interaction	Time period X Sex	777.413	2	1.181
Model		26262.735	5	15.953***
Residual		41816.182	127	
Total		68078.917	132	

***p<0.001

Analysis of mean femoral length by time periods revealed that sexual dimorphism in stature was retained in all time periods, with males having significantly longer femurs than females (Table 4.62).

Table 4.62. ANOVA Results for Sex Differences (Males/Females) in Femur Length

	Source	Sum of Squares	df	F
Middle	Between sexes	6582.400	2	18.069***
	Within sexes	4553.600	25	
	Total	11136.000	27	
Late I	Between sexes	14634.156	1	41.453***
	Within sexes	22240.859	63	
	Total	36875.015	64	
Late II	Between sexes	4016.667	1	10.428**
	Within sexes	15021.723	39	
	Total	19038.390	40	
Combined	Between sexes	25356.127	2	38.783***
	Within sexes	42823.843	131	
	Total	68179.970	133	

**p<0.01, ***p<0.001

For assessment of correlation of stature and longevity, femur length was examined by mean age at death. Table 4.63 presents the arithmetic means calculated for

age at death and femoral length by temporal periods and sex. Table 4.64 shows the results of bivariate testing of correlation between mean age at death, femoral length, temporal periods, and sex.

Table 4.63. Comparison of Mean Age at Death and Mean Femoral Length

Sex	Time period		Femoral length	Mean age at death (in years)
Male	Middle	Mean	442.27	35.64
		N	15	14
		SD	14.40	7.17
	Late I	Mean	439.5	33.71
		N	26	24
		SD	19.82	7.35
	Late II	Mean	434.21	33.85
		N	24	24
		SD	17.71	7.50
	Combined	Mean	438.18	34.20
		N	65	62
		SD	17.94	7.29

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Sex	Time period		Femoral length	Mean age at death (in years)
Female	Middle	Mean	411.33	30.33
		N	12	12
		SD	12.25	7.41
	Late I	Mean	408.87	33.86
		N	39	36
		SD	18.08	8.66
	Late II	Mean	414.12	37.94
		N	17	16
		SD	22.10	6.57
	Combined	Mean	410.62	34.22
		N	68	64
		SD	18.21	8.25

Table 4.64. Bivariate Correlation between Mean Age at Death, Femoral Length, Temporal Periods, and Sex

		Mean age	Femur length	Sex	Time period
Mean age	Pearson Correlation	1	.021	-.670**	.045
	Sig. (1-tailed)	.	.409	.000	.230
	N	277	127	277	277
Femur length	Pearson Correlation	.021	1	-.599**	-.016
	Sig. (1-tailed)	.409	.	.000	.426
	N	127	134	134	134
Sex	Pearson Correlation	-.670**	-.599**	1	.023
	Sig. (1-tailed)	.000	.000	.	.344
	N	277	134	320	320
Time period	Pearson Correlation	.045	-.016	.023	1
	Sig. (1-tailed)	.230	.426	.344	.
	N	277	134	320	320

**p<0.01

Due to significant values showed by the bivariate test for correlations between sex differences in mean age at death and femoral length, longevity was examined for each sex separately. For further analyses femoral length was turned into z-scores (Table 4.65).

From both sexes, only males demonstrated significant correlation of age at death with femoral length. When all males were included in the analyses, taller males had a higher mean age at death than shorter males in the Late II period (Table 4.66). When, for in-depth investigation, only males with a z-score of 1.0 above and below the average were selected, taller males showed a higher mean age at death than shorter males for the sample including combined time periods (Table 4.67).

Table 4.65. Mean Age at Death and Z-Score Values of Femoral Length

Sex	Time period	Z-score above the average		Z-score below the average	
		Femoral length	Mean age at death	Femoral length	Mean age at death
Male	Middle	450.88	36.50	429.33	34.50
	Late I	458.91	32.20	425.27	34.78
	Late II	451.55	37.61	423.80	31.60
	Combined	454.14	35.28	425.33	33.37
	Combined	465.00	35.00	412.00	28.70
Female	Middle	423.40	29.20	402.71	31.14
	Late I	424.76	33.07	396.59	34.36
	Late II	436.29	37.00	398.60	38.50
	Combined	427.31	33.24	398.21	34.85
	Combined	440.00	35.10	385.27	34.81

Table 4.66. ANOVA Results for Higher Mean Age at Death of Taller Males Compared to Shorter Males (Late II)

	Source	Sum of Squares	df	F
Mean age	Between groups	203.251	1	4.100†
	Within group	1090.489	22	
	Total	1293.740	23	

†0.10<p<0.05

Table 4.67. ANOVA Results for Higher Mean Age at Death of Tallest Compared to Shortest Males

	Source	Sum of Squares	Df	F
Mean age	Between groups	216.491	1	4.726*
	Within group	916.100	20	
	Total	1132.591	21	

*p<0.05

Association between the Stress Variables

Table 4.68 presents the results of Pearson tests for correlation between enamel hypoplasia, porotic hyperostosis, cribra orbitalia, and periostitis, demonstrating strong association between stresses of cribra orbitalia and periostitis.

Table 4.68. Pearson Correlations between Enamel Hypoplasia, Periostitis, Cribra Orbitalia, and Porotic Hyperostosis

	Enamel hypoplasia	Periostitis	Cribra orbitalia	Porotic hyperostosis
Enamel hypoplasia	1	.074	.078	.155
Periostitis		1	.248**	.055
Cribra orbitalia			1	-.037
Porotic hyperostosis				1

**p<0.01

For further analyses of combined effects of stresses, markers that were scored as "slight" were excluded from the sample to examine the differences in mean age at death and stature between moderately to severely affected individuals versus non-affected individuals. Table 4.69 presents the arithmetical means of age at death and Table 4.70 shows means of femoral length for individuals affected with several stress markers versus individuals free from the corresponding stresses.

Table 4.69. Mean Age at Death (in Years) of Individuals Affected with Two Types of Stresses versus Non-Affected

Variable	Sex	Affected with two stresses		Non-affected	
		Mean	SD	Mean	SD
E.hypoplasia/ Cribra orbitalia	Males	17.3	1.5	26.8	8.0
	Females	20.0	4.0	28.1	9.2
E. hypoplasia/ P.hyperostosis	Males	28.6	7.2	25.8	9.3
	Females	21.1	3.8	31.7	7.4
E. hypoplasia/ Periostitis	Males	26.0	12.7	27.7	8.0
	Females	18.7	3.4	26.8	8.5

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Variable	Sex	Affected with two stresses		Non-affected	
		Mean	SD	Mean	SD
Cribra orbitalia/ Periostitis	Males	26.0	12.7	32.3	8.5
	Females	30.1	10.9	30.9	9.1
Cribra orbitalia/ P.hyperostosis	Males	28.7	8.4	31.9	9.5
	Females	28.7	8.1	32.7	6.7
P.hyperostosis/ Periostitis	Males	31.9	8.8	31.1	10.2
	Females	33.5	9.9	34.4	7.7

Table 4.70. Mean Femoral Length of Individuals Affected with Two Stress Markers vs Non-Affected

Variable	Sex	Affected with two stresses		Non-affected	
		Mean	SD	Mean	SD
E.hypoplasia/ Cribra orbitalia	Males	142.6	247.1	371.3	161.0
	Females	248.2	226.6	357.5	139.9
E. hypoplasia/ P.hyperostosis	Males	338.0	183.7	274.2	227.3
	Females	294.4	201.2	397.8	13.1
E. hypoplasia/ Periostitis	Males	439.5	30.4	376.8	154.3
	Females	208.2	240.5	362.2	137.3
Cribra orbitalia/ Periostitis	Males	nd	nd	382.9	147.2
	Females	300.4	193.9	374.8	113.8

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Variable	Sex	Affected with two stresses		Non-affected	
		Mean	SD	Mean	SD
Cribra orbitalia/ P.hyperostosis	Males	287.6	222.9	326.7	201.2
	Females	309.9	199.8	409.1	15.8
P.hyperostosis/ Periostitis	Males	218.1	233.4	312.8	208.7
	Females	260.3	214.8	408.0	17.2

Table 4.71 displays the results of an ANOVA test that showed lower mean age at death for males and females affected with both enamel hypoplasia and cribra orbitalia compared to stress-free individuals. Females affected with a combination of enamel hypoplasia and porotic hyperostosis and females affected with enamel hypoplasia and infectious disease also demonstrated a lower mean age at death than females non-affected with these stress markers (Table 4.72 and 4.73)

Table 4.71. ANOVA Results for Mean Age at Death of Individuals Affected with Enamel Hypoplasia and Cribra Orbitalia vs Non-Affected

	Source	Sum of Squares	Df	F
Males	Between groups	234.262	1	4.012†
	Within groups	1167.693	20	
	Total	1401.955	21	
Females	Between groups	251.488	1	3.561†
	Within groups	1341.750	19	
	Total	1593.238	20	

†0.10<p<0.05

Table 4.72. ANOVA Results for Mean Age at Death of Females Affected with Enamel Hypoplasia and Porotic Hyperostosis vs. Non-Affected

	Source	Sum of Squares	Df	F
Females	Between groups	445.337	1	11.710**
	Within groups	532.413	14	
	Total	977.750	15	

**p<0.01

Table 4.73. ANOVA Results for Mean Age at Death of Females Affected with Enamel Hypoplasia and Periostitis vs. Non-Affected

	Source	Sum of Squares	Df	F
Females	Between groups	208.013	1	3.281†
	Within groups	1141.188	18	
	Total	1349.200	19	

†0.10<p<0.05

Table 4.74 presents the results of ANOVA tests that showed that males affected with enamel hypoplasia and cribra orbitalia had a shorter femoral length than males free from both of these stresses. ANOVA tests also revealed lower femoral length for females affected with a combination of cribra orbitalia and porotic hyperostosis (Table 4.75).

Table 4.74. ANOVA Results for Mean Femoral Length of Males Affected with Enamel Hypoplasia and Cribra Orbitalia vs. Non-Affected

	Source	Sum of Squares	Df	F
Males	Between groups	136364.872	1	4.655*
	Within groups	615196.867	21	
	Total	751561.739	22	

*p<0.05

Table 4.75. ANOVA Results for Mean Femoral Length of Females Affected with Cribra Orbitalia and Porotic Hyperostosis vs. Non-Affected

	Source	Sum of Squares	Df	F
Females	Between groups	67265.832	1	4.501*
	Within groups	403529.409	27	
	Total	470795.241	28	

*$p<0.05$

CHAPTER 5: DISCUSSION AND SUMMARY

Discussion

Enamel Hypoplasia

Based on the data derived from the mandibular canine as the most hypoplastic tooth, around one-third of the population (36.9%) exhibited enamel defects. This prevalence is comparable to other Native American prehistoric populations with a hunter-gatherer subsistence pattern (Bartelink 2006; Goodman et al. 1980). The study revealed no differences in prevalence of enamel hypoplasia in the Ala-329 sample during three time periods.

Still, there were several results in the study, which attained only marginally significance, but might deserve attention. Analysis by age groups showed a prevalence of enamel defects in a subadult group as compared to adults in the Middle period and an increase in frequency of enamel hypoplasia in the adult group between the Middle and Late I periods. The lower prevalence of hypoplasia in subadults in other temporal periods and the fact that an increase in hypoplasia was found only among adults might suggest either a reduction in stress load on subadults in later periods or, on the contrary, an increase in stress load with a frequent and rapid mortality outcome, which would result in a lesser number of skeletal stress markers. Demographic analysis of the prehistoric populations from the neighboring Plains Miwok ethnographic area suggested a better survivorship over time of subadults after the age of 4 (Doran 1980). At the

moment, marginally significant results and a lack of a comprehensive demographic analysis of studied populations require a conservative treatment of temporal differences by age groups.

In general, the results are comparable to Bartelink's (2006) study of enamel hypoplasia on the San Francisco Bay area sample, which found no significant differences between the Middle and Late period (the Late period, however, was not separated into phases in his study). In contrast, the results from the nearby regions of Sacramento-San Joaquin Valley demonstrated increase in enamel hypoplasia from the Middle to Late I and Late II periods (Bartelink 2006; Dickel et al. 1984). Significant increase in prevalence of enamel defects was also found in Southern California (Santa Barbara) in the Late Middle period (which roughly corresponds to the Late Middle and Late I period in the Central California) (Walker and Lambert 1989).

Elevation in prevalence of enamel defects in other regions was attributed to increased stress load related to depression of food resources (Sacramento Valley), or to decrease of food resources due to climatic instability, and sanitation problems due to aggregation of people in large villages (Santa Barbara) (Bartelink 2006; Walker and Lambert 1989). Assuming that the relatively small subsamples used in this study are representative, absence of significant changes in the San Francisco Bay area can be seen as suggestive of successful adaptation of the local population to increase in population size, density, and changing environmental conditions. A beneficial geographical location, which allowed access to variety of food resources, might possibly mitigate amplitude of stresses in the San Francisco Bay area.

No statistically significant differences were observed between involvement of males and females in any of the temporal periods and in the total sample of combined periods. Because enamel hypoplasia is a marker of physiological stresses that occur during tooth formation, absence of significant differences in enamel defects suggests similar health status of both sexes during childhood. For both sexes, however, childhood stresses that resulted in enamel hypoplasia had long-term consequences for health. Individuals who had earlier onset of enamel hypoplasia were at significantly higher risk to die earlier. This finding corresponds to the results from other anthropological studies that also found correlation between childhood stresses and longevity (Goodman and Armelagos 1988; Palubeckaite et al. 2002; Slaus 2000; Stodder 1997). Explaining this phenomenon, Goodman and Armelagos (1988) speculated that childhood stresses expressed in enamel defects bring "biological damage" to individuals weakening their ability to respond to other stresses that occur during the life span. In accordance, analysis of mean age at death showed that individuals with a greater number of enamel stress markers had significantly shorter life span than those who had no enamel defects. Owing to a high rate of attrition in the Ala-329 population, mean age at death in the available sample was biased toward younger ages. Still, the applied method of recording hypoplasia, with control for attrition, makes all internal results for this stress marker comparable within the present study.

Porotic Hyperostosis

No significant temporal changes were found in prevalence of porotic hyperostosis and cribra orbitalia when all sexes and ages were considered together. Still, an increase in cribra orbitalia was observed in the adult group (mostly in females) between the Middle and Late I period, suggesting an increase in nutritionally induced stresses. Comparison with other studies of porotic hyperostosis from the region and neighboring areas presented diverse results. Bartelink's (2006) study, performed for prehistoric populations from several San Francisco Bay area sites also did not reveal differences in prevalence of porotic hyperostosis between time periods. In contrast, the results from Sacramento Valley showed significant increase in porotic lesions in the Late period (Bartelink 2006).

There were no statistically significant differences observed between sexes in prevalence of porotic hyperostosis. When controlled for sex and time periods, however, the subsample of males demonstrated a decrease in porotic hyperostosis between the Late I and Late II periods. In contrast, cribra orbitalia was more prevalent in females, specifically in the Late II period. Although preferential treatment of boys over girls in later periods is a possible hypothesis to explain the observed trend, no ethnographic information is available in this regard. Observed variation may be attributed to differences in dietary practices of males compared to females. Traditional difference in access to various food resources based on sexual division of labor (males are hunters, females are gatherers) was probably strengthened in later temporal period due to increased complexity in the organizational structure of the society. Hildebrandt and

McGuire (2002) suggested, for instance, that hunting might mean more than subsistence strategy for males and was probably tied to symbolic aspect of culture related to gender-differentiated expression of prestige, kinship pattern, and political authority. Higher carbon and nitrogen collagen isotope signatures in males compared to females in the Late period suggest that males from the bay area had a greater access to enriched marine protein than females (Bartelink 2006). At the same time, prevalence of anemia among females and persistence of the cribratic lesions into adulthood can be attributed to loss of iron and other nutrients due to menstruation, childbearing, and lactation. Based on Doran's (1980) observation of the highest rate of population growth in the Late period and subsequent decrease in birth spacing, it is possible to infer increase in childbearing stresses in females.

Although anemia can persist or be acquired in adulthood, the formation of porotic lesions on the bones occurs only in childhood, due to bone plasticity (Stuart-Macadam 1985). Predilection of active porotic lesions within the subadult group correlates with this phenomenon. While cribra orbitalia did not show significantly greater prevalence in any of the age groups, porotic hyperostosis was more prevalent among adults, suggesting that numerous individuals survived this type of childhood stress in all time periods.

Analysis of mean age at death showed that childhood stresses that resulted in porotic hyperostosis had an adverse effect on longevity. Individuals affected with porotic hyperostosis and cribra orbitalia showed lower mean age at death than non-affected individuals. Similarly, for males specifically, those affected with cribra orbitalia showed an average lower longevity compared to non-affected males. The adverse affect of

anemia on longevity was observed in other anthropological studies (Blom et al. 2005; Mittler and Van Gerven 1994). In contrast, mean age at death of the female group shows little correlation with porotic stresses, when lesions for all levels of severity were included. Females demonstrated a steady increase in mean age at death over time in both affected and stress-free groups. The observed sex-related divergence led to a significantly higher mean age at death of females affected with porotic stresses compared to affected males. This finding may be explained by the observation that females were possibly more buffered against environmental stresses than males, as suggested in some anthropological and clinical studies (Ortner 1998; Stinson 1985). However, knowing that Ala-329 was only partially excavated, other interpretations should be considered. Increased social stratification in the Late period and the possible differential placement of older females within certain parts of the cemetery might influence the finding.

The etiology of porotic lesions observed in the examined population can be attributed to various pathologies. Porotic hyperostosis and cribra orbitalia are commonly attributed to several factors, such as iron deficiency anemia (Stuart-Macadam 1985), megaloblastic anemia due to folic acid deficiency (Fairgrieve and Molto 2000), and scurvy due to vitamin C deficiency (Brickley and Ives 2006), infestation with parasites, and chronic infectious diseases (Blom et al. 2005; Lallo et al. 1977; Walker 1986). Moreover, it should be acknowledged that the mild expression of porotic hyperostosis and cribra orbitalia that was prevalent in the present sample cannot be properly diagnosed through gross observation. Histological study by Schulz (2001) and Wapler and colleagues (2004) showed that skeletal markers, which appear as porotic hyperostosis and

cribra orbitalia on gross observation, might also be lesions resulting from inflammatory processes or osteoporosis. Because of the multietiological aspect of porotic hyperostosis and cribra orbitalia, mild, non-diagnostic forms of their expression cannot be interpreted as markers of strictly nutritional stress, but can potencially serve as markers of general stress.

Slight periosteal bone deposition undistinguished from healed porotic lesions might have influenced the recorded frequency of porotic hyperostosis (Schultz 2001). Also, chronic infectious diseases could be one of the factors leading to iron deficiency and consequent porotic hyperostosis (Lallo et al. 1977). It should be mentioned in this regard that the prevalence of markers of infectious diseases observed in the present study on postcranial bones is essentially lower than the prevalence of porotic hyperostosis and cribra orbitalia. Thus, infectious diseases can be considered as one of the possible contributory factors, but cannot fully explain the relatively high frequency of porotic lesions found in this population.

One of the common interpretations for porotic lesions in the anthropological literature is iron deficiency anemia. The best source of dietary iron is meat. Analysis of faunal remains from the bay area indicates a shift from consumption of high-ranked to low-ranked mammals occurred in the Late Holocene, suggesting a depletion of food resources (Broughton 1994). Isotope study also confirmed change in dietary practice in the Middle and Late periods, both of which demonstrated generally similar isotopic signatures, indicating a shift toward smaller prey and a higher consumption of shellfish and plant food compared to the Early period (Bartelink 2006). Despite the observed shift

in diet, Bartelink (2006) concluded that populations from the San Francisco Bay area still had an adequate amount of protein in their diet. Thus, porotic hyperostosis most probably cannot be explained by insufficient dietary intake of iron, although, again, females might be considered at a higher risk to develop anemia due to insufficient nutrient supply among the other causes due to higher dietary requirements related to childbearing.

Gastrointestinal infections from contaminated water sources accompanied by intestinal bleeding and diarrhea, which led to iron and other nutrients deficiency, might be one of the factors that influenced the frequency of porotic hyperostosis (MacPhail and Bothwell 1992). Walker (1986) noted that populations from the Channel Islands, with their limited drinking water sources and growing population, had higher prevalence of cribra orbitalia than mainland populations from the Santa Barbara region that had many freshwater springs available. Water-borne diseases acquired through contaminated water sources still present a considerable burden for human health on a global scale (Leclerc et al. 2002). Heavy use of available water sources by a growing population might possibly lead to contamination of local water sources. The present sudy, as well as the Bartelink's study (2006), which included data from several other east bay area shore sites embracing all three temporal periods, did not reveal an increase in prevalence of anemic lesions in relation to an increase in population growth. Thus, while diarrheal gastroinfections cannot be rejected as one of the contributing causative factors for prevalence of anemic lesions in the examined population, other etiological factors should also be considered.

Several studies suggested intestinal parasites as one of the etiological factors of porotic hyperostosis (Blom et al. 2005; Cybulski 1977; Reinhard 1992; Sullivan 2005; Walker 1986). Walker (1986) and Sullivan (2005) argued that fish and shellfish were the probable sources of intestinal parasites. High frequency of cribratic lesions in prehistoric populations from the Santa Barbara Channel Islands, which had a rich protein diet based on consumption of fish, led Walker (1986) to conclude that porotic hyperostosis in these populations was not due to inadequate diet, but was probably related to infestation with intestinal parasites along with diarrheal infections and subsequent loss of necessary elements. Fish tapeworms, *Diphyllobothrium spp.*, were identified in several prehistoric sites from the Pacific Coast (British Columbia) (Bathurst 2005). Consumption of fish or shellfish contaminated with this parasite might lead to development of megaloblastic anemia (or pernicious anemia in severe cases) due to loss of vitamin B12 (Rifkind et al. 1986). The population from Ala-329, as well as from the other coastal sites in the San Francisco Bay area, was highly oriented toward consumption of marine resources (Bartelink 2006). Bathurst (2005) also identified a high frequency of human roundworms, *Ascaris lumbricoides*, in some prehistoric populations from the Pacific Coast, concluding that parasite burden can contribute to occurrence of anemia in prehistoric populations. Similarly, Ubelaker (1992) suggested that parasitism, possibly related to infestation with hookworm, *Ancylostoma duodenale*, was an important casual factor of porotic hyperosotosis among prehistoric populations from several coast settlements in Ecuador. Based on the presented information, it is possible to infer that

infestation with parasites might be considered as one of the most contributive factors of anemia in the Ala-329 population.

Some considerations can be mentioned regarding the other possible factors influencing prevalence of anemic lesions in the studied population. The present study observed a significant increase in the Late I period in prevalence of cribra orbitalia for adults (mostly among females), and a marginally significant increase in active cribratic lesions and lesions of porotic hyperostosis in subadults in the Late I period. Both the Middle and Late I periods are characterized by environmental instability known as the Medieval Climatic Anomaly. The intensity of this global event varied in time, producing episodes of more severe droughts around AD 800–1350 (mostly coincident with the Late I period, according to the Central California Taxonomic System) (Ingram et al. 1996). There are no specific data, at the moment, to suggest that this warm period had an adverse effect on subsistence of indigenous people. Indirect evidence that droughts could adversely affect local environments comes from the ethnohistoric records from the Mission period. In the letter to Governor Diego de Borica, Commander Perez- Fernandez wrote in 1794 that the priests at Mission San Francisco expected more Indians joining the mission "because the pagans are without food, having lost their harvest due to the severity of the drought"(Milliken 1995:131). Anthropological studies from Central and Southern California also suggest deterioration of health during the medieval environmental instability (Lambert 1993; Walker and Lambert 1989; Weiss 2002). The present study, however, did not find sufficient data to attribute observed increase in anemic lesions in some subgroups from the sample exclusively to impacts of climatic

fluctuations on local environments and, subsequently, on health of the examined population.

Periostitis

The study did not reveal significant differences of periosteal lesions between time periods when all sexes and ages were considered together. It is possible to state that overall load of infectious diseases remained approximately similar during the Middle and Late periods. This finding correlates with the results from other studies in California that also did not find a significant difference in the prevalence of infectious disease between the Middle and Late periods (Bartelink 2006; Lambert 1993).

No sex difference in prevalence of periostitis was found, except for a difference in severity of lesions in the Late II period. In the Late II period, females had an overall higher frequency of lesions, many of which, however, were slight. Because slight manifestations of periosteal remodeling might be attributed not only to infectious stresses but also relate to varicose veins, venous statis, or recurrent minor trauma (Roberts and Manchester 1995:130), prevalence of periosteal lesions in females in the Late II period cannot be attributed strictly to prevalence in infectious diseases. At the same time, males demonstrated some prevalence of severe lesions in the Late II period. Two out of five moderate to severe infectious lesions in males from this time period are cases of possible osteomyelitis. The osteomyelitic cases might possibly be attributed to trauma because, according to Ortner and Putschar (1985), hematogenous osteomyelitis rarely occurs in adults. In accordance with severity of involvement, affected males demonstrated lower mean age at death than lesion-free males in this period. Both affected and lesion-free

females showed increase in mean age at death over time resulting in a significantly higher mean age at death of females as compared to males in the Late II period. Slight differential sex involvement is not surprising, since Damon (1964) observed that females commonly exhibit a higher morbidity, while males usually have a higher mortality rate. Once again, however, differential placement of older females within the cemetery should also be considered.

Analysis of the sample by age showed that in the Middle period the subadult group exhibited higher prevalence in periostitis than adults, while the Late period presented the reversed pattern of infectious lesions with higher rate of prevalence in adults. Anthropological studies of archaeological and contemporary populations demonstrated that children are the most sensitive to environmental and cultural conditions and, thus, represent the group that is at highest risk of increased morbidity and mortality (Goodman and Armelagos 1989; Wood 1983). It is possible to infer that the higher frequency of infectious markers among subadults versus adults in the Middle period compared to the reverse distribution in the Late period implies that subadults experienced more beneficial life conditions in the later periods. It should be remembered, however, that periosteal lesions represent a tip of the iceberg of a total load of infectious diseases in a population. Many individuals, especially children, could die before bone lesions had time to form. As such, the speculation regarding the improved life conditions for subadults in the Late period should be treated as a possible explanation, which, however, requires further examination.

Prevalence of periostitis in the adult group found in the Late period and the healed state of many lesions are suggestive of late onset of periosteal pathology and successful survivorship of the individuals exhibiting the lesions (Grauer 1993). Accordingly, the study found that individuals affected with periostitis exhibited, in average, no difference in mean age at death compared to non-affected individuals. Lack of differences in mean age at death between individuals affected and non-affected with periostitis corresponds with an observation that most of the periosteal lesions in this population were slight and, thus, did not present a serious threat to health.

Although this study did not have a goal to make diagnoses, several observations can be done regarding the character and possible etiology of recorded cases. Most commonly, periostitis and several cases of possible osteomyelitis were observed on tibiae, followed by cases on both tibiae and fibulae, and less often on tibiae and femora. The cases of osteomyelitis found among adult individuals in the sample can probably be attributed to staphylococcus infections. According to Ortner and Putschar (1985), 90% of the cases of osteomyelitis occur due to this bacterium. Traumatic origin of one out of three cases with possible osteomyelitis is probable but not confirmed.

The majority of the lesions seen in the sample are cases of mild periostitis, which represent low-grade inflammatory processes with non-specific etiology that might relate to various chronic infections and trauma (Roberts and Manchester 1995). Pierce (1982), by differential diagnosis of infectious lesions, suggested primary periostitis, pulmonary osteoarthropathy, fibrous displasia, hematogenous periostitis, and osteomyelitis among the possible diagnoses for inflammatory lesions found in the Ala-239 sample.

Another possible interpretation of some cases of chronic periostitis with predilection to the tibiae is non-venereal treponemal infection, such as yaws or endemic syphilis. Some tibiae from Ala-329 exhibited remodeling of the anterior crest producing mild appearance of saber shin. Presence of treponemal infections in Central California was suggested by Roney (1959) in his analysis of cases with slight tibial periostitis from Son-299 site. Walker and colleagues (2005) conducted a study of treponemal infections in the Santa Barbara area. Employing differential diagnosis and histological analysis, they confirmed existence of nonveneral treponemal disease in Southern California prior to European contact. Histological study of the periosteal lesions from Ala-329 is suggested for further investigation of suspected cases and determination of presence of trepanematosis in Central California.

Moreover, one of the adult individuals from the site had pathological lesions on proximal tibial joint surface of both tibias that might be consistent with tuberculosis-like or fungal infections. The lesions were circumscribed and lytic, with very little periosteal reaction on the sides of the tibial metaphyses. The affected parts of the joints and cancelous bones underneath them represent cystic-like lesions with smooth surfaces. Presence of tuberculosis in Central California was proposed by Roney (1959), who identified it in one of the skeletons from the Sonoma County coastal site dated to the Middle period (Son-299). Wu (1999) suggested diagnosis of *M. bovis*, *M. tuberculosis*, or *C. immitis* infection for six individuals from neighboring site of SCl-38, who exhibited lytic lesions on vertebrae. Knees are often reported as a site of bone involvement in tubercular and *C. immitis* infections (Aufderheide and Rodriguez-Martin 1998:139, 216).

Tentative diagnosis of coccidioidomycosis seems very possible. Specific cause of lesions observed at Ala-329 requires, of course, further examination.

Stature

Difference in femoral length between males and females was found to be significant in all three time periods and, subsequently, male and female samples were examined separately. Neither of the sexes exhibited change in femoral length over time, allowing the suggestion that environmental and cultural changes observed archaeologically in the bay area in the Middle and Late periods have not adversely influenced overall levels of physiological stress. Absence of significant changes in stature during the three temporal periods correlates with lack of significant changes in the prevalence of other analyzed stress indicators over time found in the study

Other studies of stature of the San Francisco Bay area populations revealed similar results. Bartelink (2006), in his study of stature in the San Francisco Bay area, also did not observe any changes in femoral length between the Middle and Late periods. Due to small sample size, Bartelink (2006) could not confirm his finding statistically. Ivanhoe and Chu (1996) also observed no changes in cranioskeletal size between the Middle and Late Phase I periods, but were able to detect a slight decline between the Phases I and II of the Late period. As Ivanhoe and Chu (1996) admitted, however, decline between the Phase I and Phase II of the Late period was not significant for femur and tibia parameters and only marginally significant for cranial capacity and partial skeletal parameters. Thus, several studies, including the present work, performed on the

San Francisco Bay area samples are in agreement regarding the absence of significant changes in stature in the region in the Middle and Late periods.

In contrast, the studies of stature in other regions of California found significant decline of stature over time (Ivanhoe 1995; Lambert 1993). Lambert (1993) reported a gradual decrease of femoral length in populations from the Santa Barbara area from the Early to Late period with overall loss of about 10 cm of individual stature. Decrease in femoral length in the Santa Barbara area was related to overall health decline observed in the study as inferred from increased prevalence of markers of infectious diseases. Steady decline of stature from the Early to Late period was also observed in the Sacramento Valley (Ivanhoe 1995). Ivanhoe (1995) attributed decrease in cranioskeletal size of populations from the Sacramento Valley to increased reliance on acorn as a staple of diet and increased demographic stress in females, which led to calcium deficit in the body impeding the process of normal growth.

Analysis of mean age at death showed that males demonstrated positive correlation between stature and average life span. Taller males exhibited greater mean age at death than shorter males. Because stature serves as an indicator of the overall load of physiological stress during the period of childhood growth and development, it can be interpreted that shorter individuals had a higher stress load. Similar correlation of shorter stature with shorter longevity was found in other anthropological studies, which showed that environmental influences that occur during the childhood growth period not only determine an individual's height, but also affect an individual's health and longevity later in life (Gunnel et al. 2001; Kemkes-Grottenthaler 2005). Females did not demonstrate a

correlation between mean age at death and stature probably because they are biologically more buffered against adverse environmental influences. On the other hand, the possibility of population mix might be considered. Although there are not enough archaeological and bioarchaeological studies to infer population interbreeding, marriage of females from other groups with resident males from Ala-329 might be a possible scenario. In such a case, female stature cannot serve as an indicator of physiological stress within a single locale. At the moment, further studies on population homogeneity are suggested.

Correlation between Variables

The highest correlation between combined variables, mean age at death, and femoral length was found for combinations of enamel hypoplasia and lesions of cribra orbitalia and porotic hyperostosis. Enamel hypoplasia can occur as a result of short-term life threatening conditions, such as starvation or serious infectious diseases with high fever, that cause temporary cessation of enamel formation (May et al. 1993; Pindborg 1982). Anemic lesions of porotic hyperostosis and cribra orbitalia indicate insufficient nutrient supply during periods of subadult growth. Many clinical and anthropological studies suggested that inadequate nutrition or nutrient loss due to intestinal infections act as the most influential factors that interfere with normal growth and development (Bogin 1988; Scrimshaw et al. 1968; Thein-Hlaing et al. 1991). Correlation of enamel hypoplasia with anemic lesions demonstrates an adverse synergetic effect of nutritional and infectious stresses on young subadults. As a result, individuals of both sexes who experienced moderate to severe level of physiological stress concurrently in both enamel

hypoplasia and cribra orbitalia/porotic hyperostosis showed lower longevity and shorter stature than individuals free from both stresses. The high correlation of cribra orbitalia with periostitis suggests that anemic stresses in early childhood had serious long-term consequences for an individual's health, making individuals essentially more susceptible to infectious diseases. Notably, both stresses are more prevalent in females in the Late II period.

It was noted in this study that, unlike males, females did not exhibit correlation between mean age at death, stature, and skeletal markers of stress when "slight" sress markers were included in the analysis and each stress marker was considered separately. When stress indicators were combined and only moderate/severe cases were included in the analysis, females showed good correlation between the chosen variables. This finding, as it was suggested earlier, is possible to attribute to a phenomenon observed in other studies that females might be better buffered than males from environmental stresses (Ortner 1998; Stinson 1985).

Summary

The present study examined health of the prehistoric population, which occupied the eastern San Francisco Bay area site, CA-Ala-329, for approximately 2000 years. Based on the archaeological data that indicated population growth, density, and depression of food resources in the Late Holocene, the hypothesis regarding decline in health during the three time periods, Middle, Late I, and Late II, was tested. The results

of the study showed that prevalence of observed osseous markers of stress did not differ consistently between time periods. Consequently, sufficient evidence does not exist to demonstrate a clear health decline through time leading to refutation of the proposed hypotheses. Stature did not change significantly, confirming no dramatic change in adequacy of nutrition and overall health during observed time periods. Thus, it is possible to suggest that analysis of prevalence of stress markers during the time span of approximately 2000 years indicates an overall successful adaptation of the studied population to changing environmental and cultural conditions.

In parallel to the hypothesis, the study also investigated three research questions. The first question was whether the cultural and environmental changes differentially affected males and females of the examined populations. This study did not detect significant differences in prevalence of stress markers between males and females, except for prevalence in anemic and slight periosteal lesions in females in the Late II period. The latter could be possibly explained by difference in dietary practice between males and females and decrease in spacing between childbearing for females. Still, the study observed a gradual increase in mean age at death in females over time, which resulted in a higher mean age at death in females compared to males in the Late II period. This finding is possible to attribute to differential placement of older females within the cemetery in the Late period. Further investigation with a larger sample is suggested to clarify the trend observed for females in the Late II period.

The second question was whether the pattern of stress distribution between adult and subadult segments of the population changed over time. For the adult group, analysis

of the distribution of stress load by age group revealed an increase in prevalence of cribra orbitalia and periostits in the Late II period (as seen in females). No significant changes over time in the prevalence of anemic stresses for subadults were found. Lower prevalence of stress markers of enamel hypoplasia and periostiteal in the Late periods in subadults, the most sensitive demographic group to environmental and cultural influences, might be indicative of improvement in living conditions over time for this age group. However, because decrease in markers of infectious diseases may also mean an increase in mortality without stress markers on bones and because results attained only marginal significance, this hypothesis requires further examination with reconstruction of a demographic profile of the populations from the area by time periods.

The third question was whether an individual's longevity affected by compromised health is correlated with the observed stress markers. Both males and females demonstrated a positive correlation between stress markers, femoral length, and mean age at death, showing greater life span and taller stature in stress-free than stress loaded individuals. Combination of stresses as indicated by enamel hypoplasia and anemic lesions produced the most adverse effects on mean age at death and stature of affected individuals of both sexes. Males, on average, demonstrated correlation between stresses and mean age at death even when slight cases were included. Females showed correlation between mean age at death, stature, and stress when only moderate/severe cases were included, presumably exhibiting higher natural buffering from environmental stresses than males.

Limitation of the Present Study and Suggestions for Further Research

Despite the substantial size of the collection under the study, the examined sample cannot be considered representative of the population that inhabited the area in prehistoric time. First of all, only a small segment of the mound was excavated. Secondly, some skeletal remains were damaged by taphonomic processes, perturbation, and excavation. Subdivision of sample by time period, sex, and age group further decreased the sizes of sub-samples, influencing reliability of the results and ability to perform some statistical and demographic analyses. Moreover, the dating of the sample is not sufficiently reliable. The burials were assigned to temporal periods based on analysis of accompanied assemblage of artifacts, depth of the burial within the mound, and small percentage of radiocarbon dates.

Based on the observations made in this study some recommendations for future research can be suggested. This work demonstrated that in assessment of health of archaeological population, the use of several skeletal markers helps in determining the etiology of stresses and their interrelationship. Combination of various stress indicators is recommended to use in future studies to distinguish nutritional stresses from stresses related to infectious diseases. To continue investigation of health pattern in the San Francisco Bay area, larger sample size has to be used to increase reliability of the results and to allow more fine-grained analysis of stresses by age groups and sexes. For further understanding of pattern of health in the region, comprehensive demographic analysis is recommended to be performed separately for males and females by temporal periods.

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